"Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard"

Thesis submitted to the Faculty of

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

For the award of the degree of MD/MS in the Subject of

Obstetrics & Gynaecology

By

Dr. Sanjay Sonkamble,

(MH2021/6480)

Under the guidance of

Dr. Ajay Halder,

Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

COPYRIGHT DECLARATION

We hereby declare that **ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL**, shall have the rights preserve, use or disseminate this thesis in any format for academic/research purposes.

Dr. Sanjay Sonkamble,

Junior Resident (Academic),

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Guide:

Dr. Ajay Halder,

Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

डॉ.अजय हल्दर / Dr. AJAY HALDER
Reg.No.MCI-6504 M.S. (Obst & Gynae) D.N.B.
प्राध्यापक / Professor
प्रसूति एवं स्त्री रोग विभाग / Dept. of Obstetrics & Gynaecology
अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल-462020
All India Institute of Medical Sciences Bhopal-462020

Co-Guide(s):

Dr. Shweta Patel,

Associate Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

DR. SHWETA PATEL
Reg.No.MP-6661 MBBS,MD,FMAS
Associate Professor
Dept. of Obstetrics & Gynaecology
AIIMS,Bhopal-462020 (M.P.)

Dr. Ujjwal Khurana,

Associate Professor,

Dept. of Pathology & Lab Medicine,

AIIMS, Bhopal

डॉ. उज्ज्वल खुराना / Dr. Ujjawal Khurana
M.B.B.S., M.D., D.N.B.
सह प्राध्यापक / Associate Professor
पेथोलॉजी एवं लैब मेडिसिन विभाग
Department of Pathology & Lab Medicine
अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल-462020
All India Institute of Medical Sciences Bhopal-462020

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

UNDERTAKING

I, Dr. Sanjay Sonkamble, MD/MS Scholar registered in the batch (Month, Year), vide registration no. (Number), hereby declare that the data submitted for my MD/MS thesis belongs to me and is original. I also certify that the work was done by me to carry out the study. The institute shall not be responsible for any dispute(s), or conflict(s) of interest(s) that may arise out of this thesis at any stage.

Dr. Sanjay Sonkamble,

Junior Resident (Academic),

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Guide:

Dr. Ajay Halder,

Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

डॉ. अजय हल्दर / Dr. AJAY HALDER
Reg. No. MCI-6504 M.S. (Obst & Gynae) D.N.B.
प्राध्यापक / Professor
प्रसूति एवं स्त्री रोग विभाग / Dept. of Obstetrics & Gynaecology
All India Institute of Medical Sciences Bhopal-462020

Co-Guide(s):

Dr. Shweta Patel,

Associate Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Dr. SHWETA PATEL
Reg. No. MP-8661 MBBS MD,FMAS
Associate Professor
Dept. of Obstetrics & Gynaecology
AIIMS Bhopal-462020

Dr. Ujjwal Khurana,

Associate Professor,

Dept. of Pathology & Lab Medicine,

AIIMS, Bhopal

डॉ. उज्ज्वल खुराना / Dr. Ujjawal Khurana
M B B S M D D N B
सह प्राध्यापक / Associate Professor
पेथोलॉजी एवं लैब मेडिसिन विभाग
Department of Pathology & Lab Medicine
अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल-462020
All India Institute of Medical Sciences Bhopal-462020

Place: Bhopal

Date:

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

CERTIFICATE

This is to certify that the thesis entitled, "**Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard**", is a bonafide and original work carried out by **Dr. Sanjay Sonkamble**, under the guidance of **Dr. Ajay Halder**, Professor, Dept. of Obstetrics & Gynaecology.

Guide:

Dr. Ajay Halder,
Professor,
Dept. of Obstetrics & Gynaecology,
AIIMS, Bhopal

डॉ.अजय हल्दर/Dr.AJAY HALDER M.S. (Obst & Gynae) D.N.B. Reg.No.MCI-6504 प्राध्यापक/Professor विभाग/Dept. of Obstetrics & Gynaecology अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल-462020 All India Institute of Medical Sciences Bhopal-462020

Co-Guide(s):

Dr. Shweta Patel,
Associate Professor,
Dept. of Obstetrics & Gynaecology,
AIIMS, Bhopal

Dr. SHWETA PATEL Reg. No MP-8661 MBBS,MD,FMAS Associate Professor Dept. of Obstetrics & Gynaecology AIIMS Bhopal-462020 (M.P.)

डॉ. उज्ज्वल खुराना/Dr. Ujjawal Khurana M.B.B.S., M.D., D.N.B. सह आचार्य/Associate Professor पैथोलॉजी एवं लैब मेडिसिन विभाग Department of Pathology & Lab Medicine अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल All India Institute of Medical Sciences Bhopal-462020

Dr. Ujjwal Khurana,
Associate Professor,
Dept. of Pathology & Lab Medicine,
AIIMS, Bhopal

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

CERTIFICATE

This is to certify that the thesis entitled, "**Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard**", is a bonafide and original work carried out by **Dr. Sanjay Sonkamble**, under the guidance of **Dr. Ajay Halder**, Professor, Dept. of Obstetrics & Gynaecology, and **Dr. K. Pushpalatha**, Professor & Head, Dept. of Obstetrics & Gynaecology, **Dr. Shweta Patel**, Associate Professor, Dept. of Obstetrics & Gynaecology, and **Dr. Ujjwal Khurana**, Associate Professor, Dept. of Pathology & Lab Medicine.

Dr. Sanjay Sonkamble

Junior Resident (Academic),

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Guide:

Dr. Ajay Halder,

Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

डॉ.अजय हल्दर / Dr. AJAY HALDER
Reg.No.MCI-6504 M.S. (Obst & Gynae) D.N.B.
प्राध्यापक / Professor
प्रसूति एवं स्त्री रोग विभाग / Dept. of Obstetrics & Gynaecology
अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल-462020
All India Institute of Medical Sciences Bhopal-462020

Co-Guide(s):

Dr. Shweta Patel,

Associate Professor,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Dr. SHWETA PATEL
Reg. No. MP... MBBS,MD,FMAS
...fessor
...Gynaecology
...020 (M.P.)
Dept. ... AIIMS, Bhopal-462020

डॉ. उज्ज्वल खुराना / Dr. Ujjawal Khurana
M B B S., M.D., D.N.B.
सह प्राध्यापक / Associate Professor
पैथोलॉजी एवं लैब मेडिसिन विभाग
Department of Pathology & Lab Medicine
अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल-462020
All India Institute of Medical Sciences Bhopal-462020

Dr. Ujjwal Khurana,

Associate Professor,

Dept. of Pathology & Lab Medicine,

AIIMS, Bhopal

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

CERTIFICATE

This is to certify that the thesis entitled, "**Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard**", is a bonafide and original work carried out by **Dr. Sanjay Sonkamble**, under the supervision and guidance of **Dr. Ajay Halder**, Professor, Dept. of Obstetrics & Gynaecology, AIIMS, Bhopal

K Pushpalatha 19/11/2024

(Dr. K. Pushpalatha)

Professor & Head,

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Dr. K. PUSHPALATHA MBBS,MS
Reg.No.MP-23817
Professor and Head
Department of Obstetrics & Gynaecology
AIIMS, Saket Nagar, Bhopal-462020 (M.P.)

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

CERTIFICATE

This is to certify that the thesis entitled, "**Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard**", is a bonafide and original work carried out by **Dr. Sanjay Sonkamble**, under the guidance of **Dr. Ajay Halder**, Professor, Dept. of Obstetrics & Gynaecology, AIIMS, Bhopal

Rajnish Joshi

Dean (Academics)

AIIMS, Bhopal

Place: Bhopal

Date: 15.10.2024

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

CERTIFICATE

I hereby declare that the thesis entitled, "**Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard**", is a bonafide and original work carried out by me, under the guidance of **Dr. Ajay Halder**, Professor, Dept. of Obstetrics & Gynaecology, AIIMS, Bhopal. This thesis or any part thereof, has not formed the basis of award of any degree/fellowship, or similar title, to any candidate of any University to the best of my knowledge.

Dr. Sanjay Sonkamble,

Junior Resident (Academic),

Dept. of Obstetrics & Gynaecology,

AIIMS, Bhopal

Place: Bhopal

Date: 6/11/24.

Sanjay Thesis

by sanjay

General metrics

9,988	1,432	80	5 min 43 sec	11 min 0 sec
characters	words	sentences	reading time	speaking time

Writing Issues

No issues found

Plagiarism

3 %

0 source

3% of your text matches 0 sources on the web or in archives of academic publications

ALL INDIA INSTITUTE OF MEDICAL SCIENCES, BHOPAL

Saket Nagar, Bhopal – 462020 (M.P)

Institutional Human Ethics Committee- Student Research (IHEC-SR)

www.aiimsbhopal.edu.in Email: iheepgr@aiimsbhopal.edu.in

Ref Number: AIIMS/BPL/IHECSR/July/22/PG/10 **Date:** 7th April 2023

Letter of Permission

Student Investigator:	Guide:
DR SANJAY SHRIRAM SONKAMBLE OBGY	DR AJAY HALDER OBGY Co-Guides: Dr. Shweta Patel, Dr.Ujjawal Khurana

AIIMS, Bhopal Institutional Human Ethics Committee- Student Research (IHEC-SR) in its 7th meeting held on 22nd February 2023 discussed your proposal titled **"DIGNOSTIC EFFICACY OF MODIFIED SWEDE SCORE VERSUS REID SCORE IN PRDICTION OF PRE INVASIVE CERVICAL LESION WITH CYTOLOGICAL EXAMINATION AS GOLD STANDARD"** and evaluated it for suitability for your dissertation as required for fulfilment of MD/MS program.

Following members attended the meeting:

1. Prof. D. K. Pal, Former HoD Community Medicine, GMC, Bhopal.....Chairperson
2. Prof. Rajnish Joshi, Professor of General Medicine, AIIMS, Bhopal.....Member
3. Prof. Amit Agrawal, Professor of Neurosurgery, AIIMS, Bhopal.....Member
4. Dr. Sunita Athawale, Professor of Anatomy, AIIMS, Bhopal.....Member
5. Dr. Girish Bhatt, Addl. Professor of Paediatrics, AIIMS, Bhopal.....Member
6. Dr. Ujjawal Khurana, Assoc. Professor of Pathology, AIIMS, Bhopal.....Member
7. Dr. Ankur Joshi, Assoc. Professor of CFM, AIIMS, Bhopal.....Member
8. Mr. Trivesh Goswami, Law Officer, AIIMS Bhopal,.....Member
9. Dr. Shashank Purwar, Professor of Microbiology, AIIMS, Bhopal.....Member Secretary

The decision of the committee is as follows:

The AIIMS, Bhopal IHEC-SR approves your research protocol. All research related activities must begin only after the date of this approval. The validity of this approval is till two years from the date of issuance. Please submit a closure report / renewal request before expiration of the validity for this approval.

Shashank Purwar

Dr. Shashank Purwar
Member Secretary, AIIMS Bhopal IHEC-SR
Professor of Microbiology
AIIMS, Bhopal
Contact Number: 7773010099

Table of Contents

S. No.	Section	Page Number
1	Acknowledgements	2
2	Index for Tables	3
3	Index for Figures/Plots	4
4	Index for Images	5
5	Abbreviations	6-7
6	Title	8
7	Introduction	8
8	Review of Literature	9-24
9	Aim(s)	25
10	Objective(s)	25
11	Methods	26-45
12	Results	45-63
13	Discussion	63-66
14	Limitations	66
15	Future Prospects	67
16	Conclusion	68
17	References	69-75

Acknowledgements

I would like to express my deepest and sincerest gratitude to all those who have helped me in the successful completion of my MD thesis. I would like to begin by thanking my respected guide, Dr. Ajay Halder, Professor, Department of Obstetrics & Gynaecology, for his encouragement and support. I am immensely honored to write my MD thesis under his guidance. I would like to thank Dr. K. Pushpalatha, Professor & Head, Department of Obstetrics & Gynaecology, for providing her valuable insights regarding the subject and research methodology, which have helped immensely in making this MD thesis a successful venture.

It would not have been possible to write my thesis without my co-guides Dr. Shweta Patel, Associate Professor, Dept. of Obstetrics & Gynaecology and Dr. Ujjwal Khurana, Associate Professor, Dept. of Pathology. I express my sincerest gratitude to them for their knowledge and guidance, which has helped in the completion of this task with ease.

I would also like to acknowledge all the faculty members of the Department of Obstetrics & Gynaecology, AIIMS Bhopal for their constant support and encouragement and my seniors and colleagues for their inspiring words and suggestion.

Last but not the least I would like to thank my parents and friends for their infallible support and words of inspiration.

Dr. Sanjay Sonkamble

Index for Tables

S. No.	Table	Page No.
1	Table 1- Descriptive Statistics of Age by Pap Smear Results	45
2	Table 2- Frequencies of Parity and Pap Smear Results	46
3	Table 3- Frequencies of Contraception Use and Pap Smear Results	47
4	Table 4- Frequencies of History of Sexually Transmitted Diseases and Pap Smear Results	48
5	Table 5- Frequencies of Family History of Cervical Cancer and Pap Smear Results	49
6	Table 6- Frequencies of Pap Smear Results	49
7	Table 7- Scale: Reid's Index Metrics and Performance Indicators	51
8	Table 8- Scale: Swede's Score Metrics and Performance Indicators	52
9	Table 9- AUC Comparison for Reid's Index and Swede's Scores with Statistical Analysis	54
10	Table 10- Comparison of AUC Differences Between Reid's Index and Swede's Scores with Statistical Significance	55
11	Table 11- Decision Test Statistics of Pap Smear Results with cut-off for Reid’s Index at 4	55
12	Table 12- Chi-Squared Tests for Pap Smear Results and Reid's Index Assessment	56
13	Table 13- Comparative Measures: Odds Ratio with 95% Confidence Intervals	56
14	Table 14- Test Accuracy Metrics for Reid’s Index	57
15	Table 15- Test Accuracy Ratios and Performance Metrics for Reid's Index	57
16	Table 16- Decision Test Statistics of Pap Smear Results with cut-off for Swede’s Score at 5	59
17	Table 17- Chi-Squared Tests for Pap Smear Results and Swede's Score Assessment	59
18	Table 18- Comparative Measures: Odds Ratio with 95% Confidence Intervals (Haldane-Anscombe Correction Applied)	59
19	Table 19- Test Accuracy Metrics for Swede's Score	60
20	Table 20- Test Accuracy Ratios and Performance Metrics for Swede's Score	60
21	Table 21- Cohen's Kappa Values and Confidence Intervals for Inter-Rater Agreement in Pap Smear Assessments	62
22	Table 22- Fleiss' Kappa Values and Confidence Intervals for Inter-Rater Agreement in Pap Smear Assessments	62

Index for Figures

S. No.	Table	Page No.
1	Fig 1- Box & Whisker Plot of Age Distribution in Pap Smear Results	46
2	Fig 2- Bar Plot of Parity Distribution by Pap Smear Results	47
3	Fig 3- Bar Plot of Distribution of Use of Contraception by Pap Smear Results	48
4	Fig 4- Bar Plot of Distribution of history of STDs by Pap Smear Results	49
5	Fig 5- Bar Plot of Distribution of history of Cervical Cancer by Pap Smear Results	49
6	Fig 6- Bar Plot of Distribution of Pap Smear Results	50
7	Fig 7- ROC Curve Plot for Evaluating Test Performance: The curve illustrates the trade-off between sensitivity (true positive rate) and 1-specificity (false positive rate) for the Reid's Index, depicting the area under the curve (AUC) as a measure of overall accuracy, providing a visual representation of the test's ability to discriminate between positive and negative cases.	52
8	Fig 8- ROC Curve Plot for Evaluating Test Performance: The curve illustrates the trade-off between sensitivity (true positive rate) and 1-specificity (false positive rate) for the Swede's Score, depicting the area under the curve (AUC) as a measure of overall accuracy, providing a visual representation of the test's ability to discriminate between positive and negative cases.	54

Index for Images

S. No.	Table	Page No.
1	Image 1- Normal ectocervix- normal superficial eosinophilic cells. Some debris in the background. (obj. 20x)	30
2	Image 2- LSIL- eosinophilic squamous cells with a perinuclear empty cavity surrounded by cytoplasmic thickening and with moderate nuclear enlargement- typical koilocytes. (obj. 20x)	31
3	Image 3- ASCUS- Atypical squamous cells of undetermined significance	32
4	Image 4- A- Low-Grade Squamous Intraepithelial Lesion (LSIL), B- High-Grade Squamous Intraepithelial Lesion (HSIL)	33
5	Invasive SCC- pleomorphic malignant cells, isolated or in clusters, sometimes with markedly atypical nuclei or elongated nuclei. Inflammation, blood and necrosis in the background. (obj. 20x)	34
6	Image 6- Normal Cervix- After Normal Saline. The external os is small and round. The SCJ is visible at the external os. Blue Line- SCJ	35
7	Image 7- Normal Cervix- After Acetic Acid. No acetowhite area. Mature squamous epithelium with normal capillaries is seen. Blue Line- SCJ. Blue arrow- Normal capillaries	36
8	Image 8- Normal Cervix- After Lugol's Iodine. The squamous epithelium has turned brown, and the columnar epithelium has not taken up any stain	37
9	Image 9- LSIL- After Normal Saline. The SCJ is fully visible at the external os	38
10	Image 10- LSIL- After Acetic Acid. Thin acetowhite areas are seen at the 6 o'clock and 12 o'clock positions. The lesion on the posterior lip has a sharp but irregular border. The lesion on the anterior lip has an irregular border. Both lesions arise from the SCJ. A satellite lesion is seen at the 8 o'clock position. Blue line: SCJ. Yellow arrow: Acetowhite area. Green arrow: Satellite	39
11	Image 11- LSIL- After Lugol's Iodine. There is patchy iodine uptake on the lesions	40
12	Image 12- HSIL- After Lugol's Iodine. The central red area on the cervix is not always due to columnar epithelium. Metaplasia or even CIN lesions can appear as a red patch on the cervix. In this picture the SCJ seems to be on the ectocervix	41
13	Image 13- HSIL- After acetic acid. There is a large acetowhite area with a regular margin extending to almost all quadrants. The lesion at the 7 o'clock position is denser than the surrounding areas, resulting in the inner border sign. There is a small acetowhite area at the 9 o'clock position. Blue line: SCJ. Green line: Inner border sign. Yellow arrow: Thin acetowhite area. Blue arrow: Dense acetowhite area. Green arrow: Acetowhite area with geographical margin away from the transformation zone	42
14	Image 14- HSIL- After Lugol's Iodine. The acetowhite areas have not been stained with Lugol's iodine. Green arrow: Iodine-negative	43

Abbreviations

AKT - Protein Kinase B

BAD - Bcl-2 Antagonist of Cell Death

Bcl-2 - B-cell Lymphoma 2

CIN - Cervical Intraepithelial Neoplasia

CIN1 - Cervical Intraepithelial Neoplasia Grade 1

CIN2 - Cervical Intraepithelial Neoplasia Grade 2

CIN2+ - Cervical Intraepithelial Neoplasia Grade 2 and above

CIN3 - Cervical Intraepithelial Neoplasia Grade 3

DNA - Deoxyribonucleic Acid

E2 - Early 2 (HPV gene)

E6 - Early 6 (HPV oncoprotein)

E7 - Early 7 (HPV oncoprotein)

EMT - Epithelial-to-Mesenchymal Transition

EGFR - Epidermal Growth Factor Receptor

FADD - Fas-Associated Protein with Death Domain

FOXO - Forkhead Box O

GSK3β - Glycogen Synthase Kinase 3 Beta

HDAC - Histone Deacetylase

HPV - Human Papillomavirus

HIV - Human Immunodeficiency Virus

HSIL - High-Grade Squamous Intraepithelial Lesion

IL - Interleukin

IL-6 - Interleukin 6

IL-8 - Interleukin 8

LBC - Liquid-Based Cytology

LSIL - Low-Grade Squamous Intraepithelial Lesion

LKB1 - Liver Kinase B1

LMICs - Low- and Middle-Income Countries

MHC - Major Histocompatibility Complex

miRNA - MicroRNA

mTOR - Mechanistic Target of Rapamycin

MRI - Modified Reid's Index

MSCI - Modified Swede's Colposcopic Index (Swede's Score)

N/A - Not Applicable

NCD - Non-Communicable Disease

NO - Nitric Oxide

Pap - Papanicolaou

PI3K - Phosphoinositide 3-Kinase

p21 - Cyclin-Dependent Kinase Inhibitor 1

p27 - Cyclin-Dependent Kinase Inhibitor 1B

p53 - Tumor Protein 53

pRb - Retinoblastoma Protein

RCI – Reid's Colposcopic Index

ROS - Reactive Oxygen Species

RNS - Reactive Nitrogen Species

SCC - Squamous Cell Carcinoma

TGF-β - Transforming Growth Factor Beta

TIL - Tumor-Infiltrating Lymphocyte

TNF-α - Tumor Necrosis Factor Alpha

TP53 - Tumor Protein 53 (gene)

VIA - Visual Inspection with Acetic Acid

VILI - Visual Inspection with Lugol's Iodine

VEGF - Vascular Endothelial Growth Factor

WNT - Wingless/Integrated (signaling pathway)

Diagnostic Efficacy of Modified Swede's Score vs Reid's Index in prediction of Pre-Invasive Cervical Lesion with Cytological Examination as Gold Standard

Introduction

Cervical cancer continues to be a significant public health issue worldwide. This type of cancer is mainly caused by long-lasting infections with high-risk strains of human papillomavirus (HPV), especially types 16 and 18, which account for nearly 70% of cervical cancer cases globally. (Tsikouras et al., 2016)

To understand the complexities of cervical cancer, it's essential to recognize the role of pre-malignant lesions. These lesions can be detected through screening methods like the Papanicolaou (Pap) smear and HPV DNA testing. Pre-invasive lesions are classified as cervical intraepithelial neoplasia (CIN), which is divided into three grades: CIN1, CIN2, and CIN3. Each grade reflects the severity of dysplasia, with CIN1 indicating mild changes that affect only the lower third of the cervical epithelium. (Mitra et al., 2016)

CIN1 lesions are commonly associated with transient HPV infections and have a high chance of spontaneous regression, often resolving without treatment. CIN2, characterized by moderate dysplasia, affects two-thirds of the epithelial thickness. While some CIN2 lesions may regress, the risk of progression to a more severe grade is considerably higher compared to CIN1. In contrast, CIN3 signifies severe dysplasia or carcinoma in situ, where the entire thickness of the epithelium is affected. If left untreated, CIN3 lesions have a significant likelihood of progressing to invasive cervical cancer, underscoring the critical importance of early detection and prompt intervention. (Mitra et al., 2016)

The main cause of CIN and subsequent cervical cancer is persistent infection with high-risk HPV types, particularly HPV 16 and 18. These oncogenic strains interfere with normal cellular processes, resulting in genetic changes in cervical epithelial cells. Since HPV infections are often asymptomatic, routine screenings such as Pap smears and HPV testing are crucial for detecting abnormal changes before they progress to invasive disease. These screening methods act as an essential safety net, enabling the identification and effective management of pre-invasive lesions to prevent the development of cancer. (Oyouni, 2023)

Cervical cancer occurs when pre-invasive lesions penetrate the basement membrane, enabling cancerous cells to invade deeper cervical tissues. The most common type of cervical cancer is squamous cell carcinoma, which develops from the flat cells lining the cervix. A smaller proportion consists of adenocarcinomas, which originate from the glandular cells of the cervix. The pathogenesis of cervical cancer is primarily linked to persistent HPV infection, which initiates a series of genetic mutations that promote uncontrolled cellular growth. Over time, these mutations result in tissue invasion and metastasis. (Tsikouras et al., 2016)

In India, cervical cancer is the third most common cancer and the second leading cause of cancer-related deaths. It is a preventable disease due to its prolonged pre-invasive stage. With effective implementation of robust screening, early detection, and appropriate management can be achieved. Colposcopy-guided cytological smear is highly effective in identifying pre-malignant lesions. Additionally, incorporating lesion size into the Swede's Score enhances its accuracy compared to the Reid's Index.

Review of Literature

Introduction

Cervical cancer remains a pressing public health concern, primarily originating in the cervix, the lower segment of the uterus that connects to the vagina. This malignancy is predominantly caused by persistent infections with high-risk types of human papillomavirus (HPV), particularly types 16 and 18. These strains are responsible for nearly 70% of cervical cancer cases worldwide. Understanding the link between HPV and cervical cancer highlights the critical need for prevention and early detection strategies, as timely intervention can dramatically lower the incidence of this disease. (Tsikouras *et al.*, 2016)

To grasp the complexities of cervical cancer, it's vital to recognize the role of pre-malignant lesions, which reflect abnormal cellular changes in the cervix. These lesions can be identified through screening methods such as the Papanicolaou (Pap) smear and HPV DNA testing. If left untreated, these lesions can progress to invasive cancer. The categorization of these pre-invasive lesions falls under cervical intraepithelial neoplasia (CIN), which is stratified into three grades: CIN1, CIN2, and CIN3. Each category indicates the severity of dysplasia, with CIN1 representing mild changes affecting only the lower third of the cervical epithelium. (Mitra *et al.*, 2016)

CIN1 lesions are often linked to transient HPV infections and have a high likelihood of spontaneous regression, meaning they frequently resolve without intervention. This aspect underscores the importance of regular monitoring to ensure these lesions do not progress. CIN2 represents moderate dysplasia, where two-thirds of the epithelial thickness is affected. Although some CIN2 lesions may also regress, the risk of progression to more severe grade is significantly higher than with CIN1. This necessitates vigilant follow-up and management to prevent the advancement of these lesions. Conversely, CIN3 indicates severe dysplasia or carcinoma in situ, where the entire thickness of the epithelium is compromised. Untreated CIN3 lesions have a high potential to progress to invasive cervical cancer, highlighting the critical importance of early detection and prompt treatment. (Mitra *et al.*, 2016)

The primary etiological factor in the development of CIN and subsequent cervical cancer is persistent infection with high-risk HPV types, especially HPV 16 and 18. These oncogenic strains disrupt normal cellular processes, leading to genetic alterations in cervical epithelial cells. HPV infections are often asymptomatic, making routine screenings like Pap smears and HPV testing essential for identifying abnormal changes before they evolve into invasive disease. These screening methods serve as a vital safety net, allowing for the detection of pre-invasive lesions, which can be managed effectively to prevent cancer development. (Oyouni, 2023)

Cervical cancer develops when pre-invasive lesions breach the basement membrane, allowing cancerous cells to invade deeper cervical tissues. The predominant form of cervical cancer is squamous cell carcinoma, arising from the flat cells lining the cervix. A smaller percentage comprises adenocarcinomas, which originate from the glandular cells of the cervix. The pathogenesis of cervical cancer is rooted in persistent HPV infection, which triggers a cascade of genetic mutations promoting uncontrolled cellular proliferation. Over time, these mutations can lead to tissue invasion and metastasis, with potential life-threatening consequences if not identified and treated early. (Tsikouras *et al.*, 2016)

The risk factors associated with cervical cancer are diverse and multifaceted. Persistent HPV infection is undoubtedly the most significant risk factor, as it directly influences the development of pre-invasive lesions that may progress to cancer. Additional risk factors include early initiation of sexual activity, having multiple sexual partners, and behaviors that enhance HPV transmission. Smoking further complicates the risk landscape by impairing the immune system's ability to manage HPV infections, thereby increasing cancer risk. Individuals with compromised immune systems, such as those living with HIV, are particularly vulnerable due to their diminished capacity to control persistent infections. Long-term use of oral contraceptives and the absence of regular cervical screenings also amplify the risk of developing cervical cancer, illustrating the need for proactive healthcare measures. (Olusola *et al.*, 2019)

Epidemiology

Cervical cancer poses a significant public health challenge, especially for women in low- and middle-income countries (LMICs). It ranks as the fourth most common cancer among women worldwide, with approximately 570,000 new cases and 311,000 deaths reported in 2018. Countries like India are particularly affected, accounting for around 97,000 new cases and 60,000 deaths annually. This troubling situation stems from inadequate screening programs, limited access to HPV vaccinations, and broader systemic issues in healthcare infrastructure. The significant disparities in cervical cancer outcomes between high- and low-income nations highlight the need for enhanced preventive measures and healthcare resources in LMICs. (Vu et al., 2018; Huang et al., 2022)

Central to the prevalence of cervical cancer is the role of human papillomavirus (HPV) infection, which is the primary risk factor associated with the disease. Research indicates that more than 80% of sexually active women will contract genital HPV at some point. While many infections resolve on their own, persistent infections with high-risk HPV types—especially HPV 16 and 18—can lead to pre-cancerous lesions and, ultimately, invasive cervical cancer. The risk of developing cervical cancer is influenced by factors beyond HPV infection, such as early sexual activity and having multiple sexual partners. Additionally, co-infection with HIV increases the likelihood of persistent HPV infection, as the virus compromises the immune system's ability to clear HPV. (Olusola et al., 2019)

Lifestyle factors, including smoking and long-term use of oral contraceptives, also elevate the risk of cervical cancer. Women who smoke are not only more susceptible to HPV but also face a higher risk of developing cervical lesions due to carcinogenic substances in tobacco. Studies show that the risk of cervical cancer doubles for women who have used oral contraceptives for more than five years and quadruples for those who have used them for over ten years. Socioeconomic status further influences risk; women from lower-income backgrounds often have limited access to healthcare, reducing their chances of receiving preventive care like HPV vaccinations and regular screenings. (Huang et al., 2022; Li et al., 2023)

Enhancing awareness and knowledge about cervical cancer prevention is vital, particularly in India, where understanding of HPV and its link to cervical cancer is significantly lacking. By improving education on the risks associated with HPV and the benefits of vaccination, we can increase vaccine uptake and encourage regular screening practices. Initiatives aimed at

informing women about their health and available preventive measures can empower them to take proactive steps, thereby lowering the incidence of cervical cancer. (Li et al., 2023)

Despite these needs, several barriers impede practical cervical cancer prevention efforts in India and other LMICs. Cultural taboos surrounding discussions of sexual health limit education about HPV and cervical cancer screening. Many women feel uncomfortable seeking care due to the stigma surrounding sexual activity, leading to hesitance in pursuing preventive measures. Access to healthcare services is often restricted, especially in rural areas where screening programs may be lacking or underfunded. Financial constraints further complicate the situation, as the costs of preventive care—such as HPV vaccinations and diagnostic tests—can be unaffordable for many families. A comprehensive approach is necessary to tackle these barriers, focusing on improving healthcare access and promoting awareness through culturally appropriate campaigns. (Shrestha et al., 2018)

Cervical cancer continues to pose a significant public health challenge in India, ranking among the most prevalent cancers affecting women in the country. Despite a gradual decline in incidence rates over recent decades, cervical cancer remains a leading cause of cancer-related morbidity and mortality. Detailed insights into the epidemiology of cervical cancer in India reveal critical patterns and trends that warrant attention. (Buskwofie, David-West and Clare, 2020)

India accounts for a substantial burden of cervical cancer, with approximately 122,844 new cases and 67,477 deaths reported annually. This malignancy represents the second most common cancer among women aged 15 to 44. The age-standardized incidence rate (ASIR) for cervical cancer in India is notably high, estimated at 22 per 100,000 women, surpassing that of neighbouring countries such as Bangladesh (19.2), Sri Lanka (13), and Iran (2.8). Additionally, regional disparities in incidence rates are pronounced. For instance, the Aizawl District in northeastern India records the highest rate at 24.3 per 100,000, followed by Barshi (19.5) and Bangalore (18.9). HPV prevalence in India ranges from 7% to 14%, with younger women exhibiting higher rates of infection. Social factors, such as early marriage and childbearing, increase the duration of HPV exposure for many women, compounding their risk. Alarmingly, fewer than 30% of women in India undergo routine Pap smears or HPV tests, contributing to late-stage diagnoses and poorer treatment outcomes. (Sreedevi, Javed and Dinesh, 2015; Vikraman, Khanna and Dandpat, 2022)

Encouragingly, there has been a notable decline in the incidence of cervical cancer across various population-based cancer registries in India, occurring even in the absence of a comprehensive screening program. For example, in Bangalore, the incidence rate decreased from 32.4 per 100,000 in 1982 to 18.7 per 100,000 in 2009. The annual percentage decrease in incidence rates has varied between 1.3% and 3.5% across different regions, with Chennai exhibiting the most significant reduction. This trend indicates progress in awareness and possible changes in risk factors, although the overall burden remains substantial. (Sreedevi, Javed and Dinesh, 2015; Vikraman, Khanna and Dandpat, 2022)

Demographic factors play a crucial role in the epidemiology of cervical cancer in India. The peak incidence typically occurs between the ages of 55 and 59, yet a significant proportion of patients present with advanced-stage disease, which adversely impacts treatment outcomes. The levels of screening coverage are shallow, especially among older and economically disadvantaged women who are at elevated risk. Opportunistic screening rates vary dramatically

across states, with Kerala reporting coverage of 6.9% and Maharashtra only 0.006%. This disparity underscores the need for targeted interventions to increase screening accessibility. (Sreedevi, Javed and Dinesh, 2015; Vikraman, Khanna and Dandpat, 2022)

The mortality rate associated with cervical cancer in India is alarming, with an age-standardized mortality rate of 16 per 100,000 women—one of the highest globally. Late-stage diagnoses and limited access to healthcare services significantly contribute to this high mortality rate. (Sreedevi, Javed and Dinesh, 2015; Vikraman, Khanna and Dandpat, 2022)

Several risk factors are associated with the high incidence of cervical cancer in India. Human papillomavirus (HPV) infection, particularly with high-risk types such as HPV-16 and HPV-18, is the primary etiological factor, accounting for a significant majority of cervical cancer cases. HPV prevalence ranges between 7.5% and 16.9%, with HPV-16 and HPV-18 being the most commonly detected oncogenic strains. Studies have shown that HPV prevalence among women diagnosed with cervical cancer ranges from 41.7% to 96.67%. Early marriage and early onset of sexual activity contribute to increased vulnerability to HPV infection, as do high parity and inadequate genital hygiene, especially in rural populations. Furthermore, women from lower socioeconomic backgrounds face higher risks due to barriers to accessing healthcare, lower educational attainment, and limited opportunities for screening. Financial constraints also represent a significant barrier to accessing preventive services. The costs associated with HPV vaccines and diagnostic tests can be prohibitively high for many families, especially in economically disadvantaged communities. Additionally, there is a general lack of knowledge regarding cervical cancer prevention strategies among the population, with many women unaware of the importance of regular screenings and vaccinations. Addressing these gaps in knowledge and access is essential for reducing the burden of cervical cancer. (Chatterjee et al., 2016; Taneja et al., 2021)

In response to the cervical cancer epidemic, India has established a national cancer control program since 1975 aimed at reducing the burden of cancer through various initiatives. The National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Disease, and Stroke (NPCDCS) focuses on opportunistic screening, particularly in rural and urban slums. HPV vaccination programs have been advocated as a preventive measure; however, their implementation faces cost and public awareness challenges. Current screening methods include Pap smears, visual inspection with acetic acid (VIA), and HPV DNA testing. Despite the availability of these methods, screening coverage still needs to be improved. (Krishnan et al., 2021; Shukla et al., 2023)

The introduction of the HPV vaccine into India's public health initiatives represents a significant step forward in the fight against cervical cancer. The vaccine is available for girls aged 9 to 14, providing a critical opportunity to reduce future cases of cervical cancer. However, vaccine awareness remains low, and vaccine hesitancy is prevalent, particularly in rural areas where cultural beliefs may shape health behaviours. Misconceptions about the vaccine and a lack of prioritization for preventive healthcare limits its coverage. (Chatterjee et al., 2016)

Current public health initiatives in India aim to improve access to screening and vaccination services. While guidelines recommend routine Pap smears and HPV DNA testing for women aged 21 to 65, the implementation of these methods is often insufficient in many regions. Ongoing efforts focus on expanding screening coverage, particularly in underserved rural areas

where healthcare access is limited. Nonetheless, significant challenges persist. Cultural and religious barriers complicate discussions about sexual health and HPV vaccination, hindering the effectiveness of public health campaigns intended to raise awareness and increase uptake. (Rajaram and Gupta, 2021)

Looking forward, the Indian government, alongside various healthcare organizations, is committed to addressing cervical cancer through comprehensive awareness campaigns, vaccination initiatives, and improved access to screening services. Evidence suggests that widespread adoption of HPV vaccines and an increased focus on screening programs could lead to a significant decline in cervical cancer incidence over the coming decades. Global initiatives, such as the World Health Organization's goal to eliminate cervical cancer as a public health problem by 2030, further support national efforts to combat this disease. (Wilailak, Kengsakul and Kehoe, 2021)

However, considerable work remains to ensure that vaccination and screening initiatives effectively reach all segments of the population, particularly marginalized and underserved communities. Addressing social determinants of health—such as education, socioeconomic status, and access to healthcare—is crucial for minimizing existing disparities. Implementing community-based interventions, integrating cervical cancer screening into primary healthcare services, and leveraging technology—such as mobile health platforms for educational outreach—will be vital in effectively reducing the cervical cancer burden in India. By taking these comprehensive measures, India can make substantial progress toward improving health outcomes for women and decreasing the incidence of cervical cancer in the years ahead.

Pathophysiology & Molecular Mechanisms

The progression to cervical cancer is a highly intricate and multifactorial process that predominantly begins with infection by high-risk HPV types, particularly HPV 16 and 18. These oncogenic viruses preferentially infect the basal cells located in the transformation zone of the cervix, where the squamous epithelium of the exocervix converges with the columnar epithelium of the endocervix. This region is particularly susceptible to HPV due to its unique anatomical features and the frequent micro-abrasions that occur during sexual intercourse. Such abrasions facilitate viral entry through which HPV can infiltrate the basal layers of the epithelium, initiating the infection. (Araldi et al., 2018; Szymonowicz and Chen, 2020)

Upon successful entry, HPV establishes an initial infection that often remains in an episomal state, allowing for replication within the host cell nucleus without integration into the host genome. This episomal form can lead to a transient infection that the immune system may clear effectively. However, persistent infection—defined as the continuous presence of high-risk HPV types for months or years—can trigger significant dysplastic changes within the cervical epithelium. These changes are characterized as CIN, with classifications that range from CIN 1 (mild dysplasia) to CIN 2 (moderate dysplasia) and CIN 3 (severe dysplasia or carcinoma in situ). Studies have firmly established a correlation between persistent high-risk HPV infections and its persistence to an increased risk of progression from CIN lesions to invasive cervical cancer. (Araldi et al., 2018; Szymonowicz and Chen, 2020)

At the molecular level, the oncogenic mechanisms induced by high-risk HPV are intricately orchestrated by its viral oncoproteins, primarily E6 and E7. These oncoproteins disrupt critical

tumor suppressor pathways and significantly alter cellular homeostasis, ultimately facilitating the progression to cervical cancer. (Graham, 2017)

The E6 oncoprotein is particularly notorious for its affinity for the tumor suppressor protein p53, which plays a crucial role in maintaining genomic integrity and regulating cellular responses to stress. Under physiological conditions, p53 acts as a guardian of the genome by faciliating cell cycle arrest and initiating apoptosis in response to various stress signals, including DNA damage, oxidative stress, and aberrant cell growth. p53 exerts its effects by regulating the expression of target genes involved in these processes, such as p21, which inhibits cyclin-dependent kinases, and pro-apoptotic factors like BAX. (Graham, 2017; Hoppe-Seyler et al., 2018; Estêvão et al., 2019; Pal and Kundu, 2019)

When HPV E6 binds to p53, it forms a complex that recruits the cellular ubiquitin ligase E6AP (E6-associated protein). This recruitment triggers the ubiquitination of p53, a post-translational modification that marks the protein for proteasomal degradation. The subsequent proteolysis of p53 effectively diminishes its functional levels within the cell, severely impairing the cell's ability to undergo cell cycle arrest or apoptosis in response to DNA damage. This loss of p53 function creates a permissive environment for cells to proliferate uncontrollably, despite the presence of genomic lesions that would typically signal for cell death or repair. (Manzo-Merino et al., 2013)

Moreover, the degradation of p53 leads to a cascade of downstream effects that further promote oncogenesis. For instance, without functional p53, the expression of p21 is reduced, allowing unregulated activity of cyclin-dependent kinases and promoting progression through the G1/S checkpoint of the cell cycle. This unregulated entry into the S phase accelerates cellular division and increases the likelihood of accumulating additional mutations. (Li et al., 2019)

In addition to its effects on cell cycle regulation, E6 also impairs the apoptotic machinery by downregulating pro-apoptotic factors such as BAX and interfering with apoptotic signaling pathways. E6 has been shown to disrupt the function of other apoptotic regulators, including FADD (Fas-associated protein with death domain) and caspase 8, key mediators of extrinsic apoptosis pathways. By inhibiting these pathways, E6 enhances the survival of HPV-infected cells that harbor potentially oncogenic mutations, allowing them to persist and proliferate. (Hoppe-Seyler et al., 2018)

The loss of p53 function not only promotes cellular proliferation but also facilitates the accumulation of additional genetic mutations, thereby fostering an environment conducive to neoplastic transformation. The continuous unchecked division of cells can lead to genomic instability, characterized by chromosomal aberrations and aneuploidy, both of which are hallmarks of cancer progression. This genomic instability is exacerbated by the persistent expression of E6, which maintains a state of dysregulation within the cell. (Li et al., 2019)

As the infected cells proliferate, the likelihood of errors during DNA replication and repair increases, leading to further mutations that activate additional oncogenic pathways or inactivate tumor suppressor genes. The progressive accumulation of these genetic changes creates a genetic landscape that is increasingly favourable to cancer development. (Graham, 2017; Hoppe-Seyler et al., 2018)

Additionally, E6's ability to influence other cellular processes contributes to the malignant phenotype. For example, E6 has been implicated in the activation of telomerase (hTERT),

which extends the replicative lifespan of infected cells by preventing telomere shortening during cell division. This extends the time frame for potential genetic mutations to accumulate, further increasing the risk of malignant transformation. (Zhang et al., 2017)

The E7 oncoprotein plays a critical role in cervical carcinogenesis by disrupting key regulatory mechanisms of the cell cycle, primarily through its interaction with the retinoblastoma protein (pRb). pRb is a pivotal tumor suppressor that serves as a master regulator of the G1 to S phase transition in the cell cycle. Under normal physiological conditions, pRb inhibits cell cycle progression by binding to E2F transcription factors, which are crucial for the transcription of genes that promote S phase entry, including those necessary for DNA synthesis and cell division. (Hoppe-Seyler et al., 2018; Estêvão et al., 2019; Pal and Kundu, 2019)

Upon HPV infection, the E7 oncoprotein binds to pRb, effectively disrupting the pRb-E2F complex. This interaction leads to the phosphorylation and subsequent degradation of pRb, resulting in its inactivation. As a consequence, E2F transcription factors are released from this inhibitory complex and translocate to the nucleus, where they activate the transcription of target genes essential for cell cycle progression. These include cyclins (e.g., cyclin E) and other factors that drive the transition from G1 to S phase. This unregulated activation of E2F-mediated transcription promotes cellular proliferation, allowing HPV-infected cells to progress through the cell cycle without the normal regulatory checks that prevent unrestrained growth. (Caldeira, Dong and Tommasino, 2005; Tzenov et al., 2013)

In addition to its effects on pRb, E7 also interacts with and neutralizes cyclin-dependent kinase (CDK) inhibitors such as p21 and p27. These proteins are crucial for the maintenance of cell cycle checkpoints and act as brakes on cell proliferation by inhibiting the activity of cyclin-CDK complexes. By binding to these inhibitors, E7 diminishes their efficacy, further facilitating the unchecked progression of the cell cycle. This action not only accelerates the entry into the S phase but also contributes to the dysregulation of G1 checkpoint control, increasing the likelihood of replicating damaged DNA. (Funk et al., 1997)

The cumulative effects of E6 and E7 oncoproteins lead to significant disruptions in the intricate regulatory networks governing the cell cycle. E6's degradation of p53, in concert with E7's inactivation of pRb and CDK inhibitors, results in a profound loss of control over cellular proliferation. This dysregulation fosters an environment conducive to genomic instability, characterized by chromosomal aberrations and aneuploidy. The failure to properly regulate the cell cycle not only allows for rapid cellular division but also increases the risk of errors during DNA replication, leading to the accumulation of mutations. (Kim and Zhao, 2005)

Genomic instability is a hallmark of cancer progression and is driven by the inability of the cell to accurately repair DNA damage. In the context of HPV infection, the combination of E6 and E7 activity creates a scenario where the cellular machinery responsible for maintaining genomic integrity is severely compromised. This leads to further mutations that can activate oncogenes or inactivate additional tumor suppressor genes, pushing cells closer to a malignant phenotype. (Li et al., 2019)

As these infected cells continue to proliferate unchecked, the progressive accumulation of genetic and epigenetic alterations heightens the likelihood of advancing from pre-invasive lesions, to invasive cervical cancer. The combination of E6 and E7-mediated disruption of cell

cycle regulation and the resulting genomic instability constitutes a critical mechanism by which HPV facilitates carcinogenesis.

As cervical lesions progress to more advanced stages, the integration of HPV DNA into the host genome becomes increasingly prevalent, particularly in high-grade squamous intraepithelial lesions (HSIL) and invasive cervical cancers. This integration often disrupts the E2 gene, which encodes a protein that negatively regulates the transcription of E6 and E7. In the absence of functional E2, the unchecked expression of E6 and E7 exacerbates their oncogenic potential, promoting further dysregulation of host cellular processes and increasing the likelihood of malignant transformation. Importantly, HPV integration is also associated with genomic instability, as it frequently occurs at fragile genomic sites, leading to chromosomal aberrations that can induce double-strand DNA breaks. The presence of a dysfunctional p53 response exacerbates these issues, further contributing to the progression of cervical lesions. (Johansson et al., 2012; Travé and Zanier, 2016; Li et al., 2019; Jose et al., 2023)

The role of epigenetic alterations in the development of cervical cancer is a complex and critical aspect of HPV-induced carcinogenesis. HPV oncoproteins, particularly E6 and E7, can instigate profound changes in the epigenetic landscape of host cells, including alterations in DNA methylation patterns, histone modifications, and the regulation of non-coding RNAs. These changes contribute to the silencing of tumor suppressor genes and the promotion of malignancy.

DNA methylation, particularly at cytosine residues in CpG dinucleotides, is a key epigenetic modification that regulates gene expression. In cervical cancer, HPV infection is frequently associated with hypermethylation of promoter regions of critical tumor suppressor genes. For instance, the gene encoding p16/INK4a (CDKN2A), a cyclin-dependent kinase inhibitor, is often found to be hypermethylated in HPV-positive cervical lesions. This hypermethylation leads to the silencing of p16, disrupting its ability to inhibit cell cycle progression, thereby facilitating uncontrolled cellular proliferation. (von Knebel Doeberitz et al., 2012; Munger, Gwin and McLaughlin-Drubin, 2013)

Similarly, genes such as LKB1 (STK11) and DAPK1 (Death-Associated Protein Kinase 1) are also subject to hypermethylation in the context of HPV infection. The silencing of LKB1, a crucial regulator of cellular metabolism and apoptosis, further exacerbates the survival advantage of HPV-infected cells. DAPK1, which is involved in apoptosis and autophagy, when silenced, contributes to the resistance of HPV-infected cells to programmed cell death, promoting cell survival and growth. (Wingo et al., 2009; Lim et al., 2010; Zhong et al., 2012; Zeng et al., 2017)

Histone modifications play a significant role in the regulation of chromatin structure and gene expression. The HPV E7 oncoprotein has been shown to interact with histone deacetylases (HDACs), enzymes that remove acetyl groups from histones, leading to chromatin condensation and transcriptional repression. This interaction contributes to chromatin remodeling that represses the expression of genes involved in cell cycle arrest and apoptosis. For instance, the repression of pro-apoptotic genes due to altered histone acetylation can diminish the apoptotic response to cellular stress, allowing HPV-infected cells to evade normal checkpoints that would otherwise lead to cell death. (Bodily, Mehta and Laimins, 2011; Lee et al., 2013)

In addition to deacetylation, the modification of histones by methylation can also influence gene expression. The interplay between acetylation and methylation checks on histones can determine whether genes are transcriptionally active or silenced. HPV oncoproteins can alter these patterns, creating a chromatin environment that is conducive to the expression of genes promoting cellular proliferation while repressing tumor suppressor genes. (Bodily, Mehta and Laimins, 2011)

Non-coding RNAs, particularly microRNAs (miRNAs), are pivotal regulators of gene expression and play significant roles in cancer biology. HPV infection has been shown to dysregulate miRNA expression profiles, contributing to oncogenesis. For example, the upregulation of miR-21 is a well-documented alteration in HPV-associated cancers. miR-21 promotes proliferation and survival by targeting and inhibiting the expression of several tumor suppressor genes, including PTEN (Phosphatase and Tensin Homolog), a critical regulator of the PI3K/AKT signaling pathway. PTEN acts as a brake on cell proliferation, and its downregulation via miR-21 leads to enhanced cell survival and growth. (He et al., 2021; Ghafouri-Fard et al., 2022)

Furthermore, other miRNAs affected by HPV can regulate pathways involved in inflammation, apoptosis, and cellular differentiation, thereby influencing the tumor microenvironment and promoting a malignant phenotype. The ability of HPV to modulate the expression of these non-coding RNAs illustrates another layer of complexity in its oncogenic potential. (He et al., 2021; Ghafouri-Fard et al., 2022)

The phosphoinositide 3-kinase (PI3K)/AKT/mTOR pathway is activated by various extracellular signals, including growth factors and hormones. Upon activation, PI3K catalyzes the conversion of phosphatidylinositol 4,5-bisphosphate (PIP2) to phosphatidylinositol 3,4,5-trisphosphate (PIP3), which in turn activates AKT (also known as protein kinase B). Activated AKT promotes cell survival and growth by phosphorylating a variety of downstream substrates involved in metabolism, protein synthesis, and cell cycle regulation. (Bossler, Hoppe-Seyler and Hoppe-Seyler, 2019)

HPV oncoproteins E6 and E7 play significant roles in the aberrant activation of this pathway. E7, through its interactions with the retinoblastoma protein (pRb), releases E2F transcription factors, which can induce the expression of various growth factor receptors and components of the PI3K pathway. This results in enhanced signaling through PI3K, promoting AKT activation. E6 further potentiates this effect by disrupting p53-mediated signaling, a critical regulator of cell cycle and apoptosis that normally functions to limit cellular proliferation in response to DNA damage. (Bossler, Hoppe-Seyler and Hoppe-Seyler, 2019)

The activation of AKT leads to the phosphorylation of several key proteins that inhibit apoptotic pathways. For instance, AKT phosphorylates and inactivates pro-apoptotic factors such as BAD (Bcl-2 antagonist of cell death) and the Forkhead box O (FOXO) transcription factors, which regulate the expression of genes involved in apoptosis. The inactivation of these pro-apoptotic proteins allows HPV-infected cells to evade apoptosis, facilitating unregulated cell division. (Manning and Toker, 2017; Nitulescu et al., 2018)

AKT also plays a role in metabolic reprogramming, promoting glycolysis and lipid synthesis, which are critical for supporting the rapid proliferation of cancer cells. By enhancing glucose

uptake and favoring aerobic glycolysis (the Warburg effect), activated AKT contributes to the bioenergetic needs of tumor cells. (Manning and Toker, 2017; Nitulescu et al., 2018)

One of the vital roles of the PI3K/AKT/mTOR pathway is in the regulation of angiogenesis, the process by which new blood vessels form from existing ones. AKT enhances the transcription of vascular endothelial growth factor (VEGF), a key pro-angiogenic factor. The increased expression of VEGF promotes the growth of blood vessels within the tumor microenvironment, ensuring an adequate supply of nutrients and oxygen to support the expanding tumor. Moreover, activation of the mTOR pathway further amplifies VEGF production, creating a positive feedback loop that enhances angiogenesis. (Karar and Maity, 2011; Manning and Toker, 2017; Nitulescu et al., 2018; Bossler, Hoppe-Seyler and Hoppe-Seyler, 2019)

In addition to the effects of HPV oncoproteins, cervical cancer frequently exhibits mutations and amplifications in components of the PI3K/AKT/mTOR pathway, such as the PIK3CA gene, which encodes the p110α catalytic subunit of PI3K. Mutations in PIK3CA can lead to constitutive activation of the PI3K signaling pathway, independent of upstream growth factor signaling. This genetic alteration exacerbates the effects of HPV-mediated signaling, further promoting tumor growth and survival. (Bossler, Hoppe-Seyler and Hoppe-Seyler, 2019; Glaviano et al., 2023)

Additionally, other components of the pathway, such as PTEN are often inactivated in cervical cancer. PTEN serves as a negative regulator of the PI3K pathway, and its loss leads to increased levels of PIP3 and, consequently, enhanced AKT activation. This dual dysregulation—through HPV oncoproteins and somatic mutations—creates a robust signaling network that supports the hallmarks of cancer, including sustained proliferative signaling and evasion of cell death. (Nero et al., 2019; Fernandes et al., 2022)

The PI3K/AKT/mTOR pathway does not operate in isolation; it interacts with other critical signaling pathways that are also dysregulated in cervical cancer. For example, the crosstalk between the PI3K pathway and the Wnt/β-catenin signaling pathway can further enhance cellular proliferation and survival. Similarly, the Notch signaling pathway may be influenced by the PI3K/AKT/mTOR axis, particularly during the later stages of tumor progression, facilitating processes such as epithelial-to-mesenchymal transition (EMT) that are critical for invasion and metastasis. (Song et al., 2015)

The central role of the PI3K/AKT/mTOR pathway in cervical cancer progression suggests that it represents a promising therapeutic target. Several small-molecule inhibitors targeting different components of this pathway are being explored in clinical trials, with the aim of disrupting the pro-tumorigenic signaling cascades initiated by HPV (Alzahrani, 2019; Glaviano et al., 2023). Additionally, combination therapies that incorporate these inhibitors with other treatment modalities, such as immune checkpoint inhibitors or traditional chemotherapy, may enhance therapeutic efficacy and provide a more effective strategy for managing HPV-associated cervical cancer.

The Wnt/β-catenin signaling pathway plays a pivotal role in regulating various aspects of cellular behavior, including proliferation, differentiation, and tissue homeostasis. Dysregulation of this pathway is a hallmark of many cancers, including cervical cancer, where

it is frequently influenced by the oncogenic effects of high-risk HPV types, particularly through the action of viral oncoproteins E6 and E7. (Yang et al., 2018)

Under normal physiological conditions, Wnt signaling is tightly regulated. When Wnt ligands bind to their receptors (Frizzled family receptors), a cascade of events is initiated that leads to the stabilization and accumulation of β-catenin in the cytoplasm. In the absence of Wnt signaling, β-catenin is targeted for degradation through a destruction complex that includes APC (adenomatous polyposis coli), Axin, and GSK3β (glycogen synthase kinase 3 beta). Once stabilized, β-catenin translocates to the nucleus, where it interacts with TCF/LEF transcription factors to activate the expression of target genes that promote cell proliferation and survival. (Liu et al., 2022)

Oncoproteins E6 and E7 disrupt the normal regulation of the Wnt pathway. E7 is particularly implicated in promoting the nuclear accumulation of β-catenin. By disrupting the functions of pRb and other key regulatory proteins, E7 can lead to the activation of Wnt target genes even in the absence of Wnt ligand binding. This aberrant activation contributes to increased cell proliferation. (Bello et al., 2015; Donmez et al., 2022)

E6 also plays a role in modulating Wnt signaling, although its primary actions are more centered on p53 degradation. However, by promoting genomic instability and altering the cellular microenvironment, E6 indirectly supports the conditions favorable for Wnt pathway activation. Additionally, mutations or silencing of key negative regulators of the Wnt pathway, such as APC, can further enhance β-catenin signaling. Loss of APC function leads to uncontrolled β-catenin accumulation and transcriptional activation of Wnt target genes, compounding the effects of HPV oncoproteins and accelerating tumorigenesis. (Drews, Case and Vande Pol, 2019)

Dysregulated Wnt signaling is a known inducer of EMT, a process whereby epithelial cells lose their adhesive properties and acquire mesenchymal traits, facilitating invasion and metastasis. EMT is characterized by the downregulation of epithelial markers (like E-cadherin) and the upregulation of mesenchymal markers (such as N-cadherin and vimentin). HPV oncoproteins, particularly E7, can promote EMT by enhancing the expression of transcription factors like SNAIL and TWIST, which repress E-cadherin expression and promote motility. (Xue et al., 2024)

Wnt signaling is also implicated in maintaining cancer stem cell properties. The activation of Wnt target genes can promote a stem-like phenotype, characterized by enhanced self-renewal and differentiation potential. This feature contributes to treatment resistance and tumor recurrence in cervical cancer. (Liu et al., 2022)

The Notch signaling pathway is another critical regulator of cell fate decisions and plays a complex role in cervical cancer. Notch signaling influences cell differentiation, proliferation, and apoptosis, and its dysregulation can have significant implications for tumor progression. Initially, HPV oncoproteins E6 and E7 can inhibit Notch signaling, favoring keratinocyte proliferation and viral replication. The downregulation of Notch activity can lead to the expansion of progenitor cell populations, providing a favorable environment for HPV persistence and replication. However, this initial inhibition has downstream consequences, allowing for the accumulation of genetic alterations and the development of dysplastic lesions. (Rodrigues et al., 2019)

As cervical cancer progresses, Notch signaling can be reactivated, which is crucial for enhanced tumorigenicity, as it can promote EMT through the activation of transcription factors like SNAIL and ZEB. This transition is vital for tumor invasion and metastasis, allowing cancer cells to migrate from the primary tumor site to distant locations. Activated Notch can promote angiogenesis through the secretion of pro-angiogenic factors, supporting the vascularization of the tumor and enhancing its growth. Notch signaling may also play a role in immune evasion mechanisms. By altering the expression of immune checkpoints and the recruitment of immune cells, Notch can help the tumor escape immune surveillance, facilitating its progression. (Henken et al., 2012)

Chronic inflammation significantly contributes to cervical cancer development, especially in the context of persistent HPV infection. This inflammation arises from the host's immune response and involves the release of pro-inflammatory cytokines like TNF-α, IL-6, and IL-8. TNF-α activates survival pathways such as NF-κB, promoting cell proliferation and inhibiting apoptosis. IL-6 supports cervical epithelial cell proliferation and angiogenesis through the STAT3 signaling pathway, aiding malignant transformation. IL-8 plays a key role in recruiting neutrophils and other immune cells, facilitating angiogenesis essential for tumor growth. (Fernandes et al., 2015; Georgescu et al., 2018)

Chronic inflammation leads to the production of reactive oxygen species (ROS) and nitric oxide (NO), which have significant implications for genomic integrity. The overproduction of ROS during chronic inflammation can cause oxidative stress, leading to DNA damage through the formation of 8-oxoguanine and other oxidative lesions. If not repaired, these lesions can result in mutations that drive tumorigenesis. While NO can have beneficial roles in immune responses, its excessive production in an inflammatory context can lead to the generation of reactive nitrogen species (RNS) that further exacerbate DNA damage. (Fernandes et al., 2015; Georgescu et al., 2018)

HPV-infected cervical lesions evade immune surveillance through several mechanisms, allowing the virus to persist and contribute to carcinogenesis. A primary method is the downregulation of major histocompatibility complex (MHC) Class I molecules by HPV oncoproteins E5, E6, and E7, which hinders the presentation of viral antigens to cytotoxic T lymphocytes. Additionally, cervical cancer cells often upregulate programmed death-ligand 1 (PD-L1), interacting with PD-1 receptors on T cells, leading to T cell exhaustion and immune evasion. The interaction between HPV-infected cells and the immune microenvironment results in the accumulation of immunosuppressive cells, such as regulatory T cells and myeloid-derived suppressor cells, which further inhibit effective anti-tumor immunity. (del Toro-Arreola, García-Chagollán and Jave-Suárez, 2015; Ling et al., 2022)

In brief, the progression of cervical carcinogenesis is a multifaceted process initiated by HPV infection and characterized by a series of interrelated molecular events that disrupt critical cellular pathways. The action of viral oncoproteins E6 and E7 results in profound alterations in cell cycle regulation, apoptosis evasion, genomic instability, and immune evasion.

Screening

Cervical cancer screening methodologies are vital for the early identification of pre-cancerous lesions and cervical cancer, particularly in asymptomatic women. The primary objective of

these screening approaches is to detect abnormal cervical cells at an incipient stage, thereby enabling intervention before progression to malignancy occurs. A comprehensive understanding of the various screening modalities available is essential for healthcare providers to optimize early detection strategies and enhance patient outcomes.

The Papanicolaou test, commonly referred to as the Pap smear, has been a foundational element of cervical cancer screening for decades. This cytological assay involves the collection of cervical cells using a spatula or brush, which are then smeared onto a glass slide for microscopic examination. The Pap smear is designed to identify cervical intraepithelial neoplasia (CIN), a condition that can evolve into invasive cervical cancer if untreated. Current guidelines recommend that women aged 21-29 undergo Pap smears every three years, while those aged 30-65 may opt for Pap testing alone every three years or in conjunction with HPV testing (co-testing) every five years. Although effective in identifying pre-cancerous changes, the Pap smear may occasionally miss early cases of cervical cancer, particularly those involving glandular cells (Nkwabong, Laure Bessi Badjan and Sando, 2019; Gavinski and DiNardo, 2023). Deep learning has emerged as a powerful tool for automating screening processes across various medical fields, significantly enhancing the accuracy and efficiency of diagnostic tasks. By leveraging advanced neural network architectures, these models can analyze complex medical images and detect abnormalities with high precision, often surpassing traditional methods. This technology not only reduces the burden on healthcare professionals but also improves early detection rates, ultimately contributing to better patient outcomes. Its adaptability allows for integration into diverse healthcare applications, making it a vital component in modern diagnostic practices (Sompawong et al., 2019)

In parallel with the Pap smear, HPV DNA testing has become an integral component of cervical cancer screening. This test specifically identifies high-risk strains of HPV, such as types 16 and 18, which are responsible for a significant proportion of cervical cancer cases. The cervical sample is analyzed for the presence of DNA or RNA from these oncogenic HPV strains. While HPV testing demonstrates greater sensitivity than the Pap smear, it may be less effective in younger women due to the transient nature of HPV infections, which often resolve spontaneously. (Garzón-Olivares et al., 2022; Gavinski and DiNardo, 2023; Sivars et al., 2023; Ali et al., 2024)

Co-testing, which integrates both the Pap smear and HPV testing, provides a holistic approach to cervical cancer screening. By collecting cervical cells for both assays during a single procedure, healthcare providers can concurrently evaluate for abnormal cellular changes and the presence of high-risk HPV. This combined methodology has been shown to significantly enhance the detection rates of both pre-cancerous lesions and HPV infections, thus offering better outcomes against cervical cancer. Current recommendations endorse co-testing every five years for women aged 30-65, reflecting its accuracy and reliability as a screening strategy. (Costa et al., 2000; Shen-Gunther et al., 2016; Gavinski and DiNardo, 2023; Ali et al., 2024)

In resource-limited settings, where advanced diagnostic tools may not be available, two effective and affordable methods for screening are Visual Inspection with Acetic Acid (VIA) and Visual Inspection with Lugol's Iodine (VILI). Both techniques enhance the visibility of abnormal cervical tissues by applying specific chemical agents. VIA involves applying a 3-5% acetic acid solution to the cervix, which causes lesions to turn white. This procedure is straightforward, provides immediate results, and allows for quick management, making it

especially suitable for resource-limited environments. However, its effectiveness is somewhat constrained by subjective interpretation and higher rates of false positives compared to more advanced methods like HPV testing and Pap smears. In contrast, VILI uses Lugol's iodine solution to stain normal cervical cells brown or black. Areas that do not absorb the iodine indicate the presence of abnormal or precancerous tissue. VILI generally exhibits higher sensitivity than VIA and also delivers immediate results, facilitating prompt clinical decision-making. However, it too faces challenges with subjective interpretation and the possibility of false positives. Both VIA and VILI are endorsed by the World Health Organization as viable screening options in settings with limited access to laboratory resources. Overall, these methods offer practical solutions for large-scale cervical cancer screening initiatives, especially in developing countries, where they can significantly enhance early detection and improve patient outcomes. (Lee, Kang and Ju, 2016; Adsul et al., 2017; Driscoll et al., 2018; Gavinski and DiNardo, 2023)

Emerging methodologies such as HPV mRNA testing offer additional layers of specificity in cervical cancer screening by detecting the activity of high-risk HPV types at the molecular level. This approach identifies E6 and E7 mRNA, oncogenic proteins associated with cervical cancer pathogenesis. Although more costly and less widely accessible than traditional tests, HPV RNA testing provides critical insights into cervical cancer risk by focusing on viral activity rather than mere presence. (Arbyn et al., 2022; Rossi and Rebolj, 2022)

Liquid-based cytology (LBC) is another advancement in cervical cancer screening. This technique collects cervical cells in a liquid medium, which enhances cell preservation and facilitates superior analysis. LBC typically exhibits higher sensitivity and accuracy compared to conventional Pap smears, thereby reducing the likelihood of false-negative results. However, its implementation may be hindered by higher costs, which can be a barrier in low resource healthcare settings. (Phaliwong et al., 2018; Ito et al., 2020)

Colposcopy serves as a crucial follow-up procedure when abnormal results are obtained from Pap smears or HPV testing. This technique employs a colposcope—a specialized magnifying instrument—to meticulously examine the cervix after the application of acetic acid, which highlights suspicious areas. This detailed visualization aids in identifying lesions that may necessitate a biopsy for further evaluation. Although colposcopy is less invasive than some other procedures, it is predominantly employed after abnormal screening findings. Artificial Intelligence (AI)-guided digital colposcopy presents a promising solution to the challenges faced in cervical cancer screening in LMICs. By utilizing deep learning algorithms to analyse colposcopic images, AI enhances diagnostic accuracy in identifying CIN and guiding biopsy decisions. Its ability to provide real-time analysis reduces false negatives and positives, improving clinical workflows and patient management. Additionally, cloud-based platforms offer remote access to expert diagnostic tools, making advanced technology available in rural areas with limited trained professionals. AI also functions as an educational resource, providing real-time guidance to novice colposcopists and helping them build their skills and confidence. AI can improve diagnostic sensitivity and specificity, effectively addressing gaps in current screening practices. (Burness, Schroeder and Warren, 2020; Xue, Ng and Qiao, 2020)

Use of Modified Swede Score and Modified Ried Index

The Modified Swede Colposcopic Index (MSCI) and Modified Reid Index (MRI) are pivotal advancements in the field of colposcopy, serving as refined tools for evaluating cervical lesions and enhancing the detection of CIN and other lesions. These indices provide a structured framework for interpreting colposcopic findings, which can be inherently subjective, thus enabling clinicians to make more informed decisions regarding biopsies and treatments. The standardization of scoring through these indices aids in improving consistency in clinical practice and ultimately contributes to better patient outcomes in cervical cancer prevention and treatment. (Bowring et al., 2010; Aue-Aungkul and Suprasert, 2015)

The MRI is an evolution of the Reid Colposcopic Index (RCI), designed to enhance the reliability of colposcopic diagnosis by refining its components. One of the key modifications involves the exclusion of iodine staining, which has been found to yield variable results in terms of its efficacy for distinguishing high-grade lesions from benign changes. Instead, the MRI retains focus on more consistently predictive features. The assessment of color plays a central role, as the acetowhite effect produced by acetic acid application is a reliable indicator of epithelial abnormalities. In this context, the degree of whiteness is classified, with opaque and dull white appearances strongly associated with higher-grade lesions (CIN2+). This contrasts with shinier, translucent white lesions, which often indicate lower-grade conditions. By establishing a clear relationship between lesion color and grade, the MRI allows clinicians to make more confident diagnoses based on observable characteristics. (Boonlikit, 2016)

The margin and surface features of cervical lesions are also essential elements of the MRI. Sharp, rolled, or irregular margins, along with changes in surface contour, can suggest a higher likelihood of CIN. In contrast, smooth, regular margins are more indicative of benign or low-grade lesions. Furthermore, the vascular patterns present in the lesions—specifically the presence of coarse or irregular vessels—serve as another critical diagnostic feature. The observation of punctation or mosaic patterns can indicate a greater likelihood of high-grade disease. (Hong et al., 2010)

While the MRI has established itself as a useful tool in clinical practice, the MSCI builds upon its strengths by incorporating additional criteria that have been shown to enhance diagnostic accuracy. One of the most significant innovations of the MSCI is the inclusion of lesion size as a key determinant of disease severity. Studies indicate that larger lesions are more likely to harbor high-grade lesions, making this factor crucial for accurate diagnosis. The MSCI emphasises a focus on lesion location and quadrant involvement, which simplifies the assessment process and allows for a more straightforward application in clinical settings. By evaluating whether a lesion spans multiple quadrants or exceeds a specific size threshold (e.g., greater than 15 mm), clinicians can better gauge the likelihood of significant pathology. (Rahman, Yadav and Tripathi, 2020)

The characteristics of acetowhite uptake are similarly examined within the MSCI framework. Dense, opaque staining following the application of acetic acid is suggestive of high-grade lesions (CIN2+), whereas lighter, transparent uptake typically indicates low-grade changes. This nuanced approach allows for a clearer differentiation between lesion types, enabling more accurate decisions. Additionally, the MSCI continues to emphasize the importance of margin and surface characteristics, whereby irregular or jagged margins, along with marked surface elevation, are indicative of a higher probability of severe lesions. (Alan et al., 2020)

The MSCI's diagnostic strengths are further enhanced by its flexible approach, allowing clinicians to adapt their scoring based on clinical objectives. For example, lower cutoffs may be employed for initial screening purposes, maximizing sensitivity and ensuring that high-grade lesions are not overlooked. Conversely, higher cutoffs can be used for confirming the presence of significant disease. This adaptability makes the MSCI particularly useful in diverse clinical contexts, accommodating various patient populations and clinical scenarios. (Priya et al., 2024)

The practicality of the MSCI is further underscored by its simplified criteria. The favor of more clinically relevant assessments like lesion location and size, streamlines the scoring process, making it easier for healthcare providers to implement in routine practice. This simplification is particularly beneficial in low-resource settings, where efficient cervical cancer screening is essential. The ease of use associated with the MSCI allows for greater accessibility to effective screening tools, potentially leading to improved detection rates. (Ranga et al., 2017)

The performance metrics of these two indices reveal the MSCI's superior diagnostic capabilities. For instance, at a threshold score of 5, the MSCI achieves an impressive 100% sensitivity for detecting CIN2+ lesions, with a specificity of 88.4%. This makes it an excellent screening tool for identifying high-grade abnormalities. When the threshold is increased to 8, the specificity improves to 90%, effectively aiding in the confirmation of significant lesions and informing treatment decisions. In comparison, the MRI achieves a sensitivity of 83.7% and a specificity of 89.7% at a threshold of 5, indicating its reliability, albeit with slightly lower precision than the MSCI. When assessed at a higher threshold of 7, the MRI does reach a specificity of 99.1%, but this comes at the cost of significantly reduced sensitivity (46.5%), rendering it less suitable for initial screening while still useful for ruling out low-grade lesions. (Ranga et al., 2017; Rahman, Yadav and Tripathi, 2020; Suwanthananon and Inthasorn, 2020; Priya et al., 2024)

In summary, the MSCI and MRI represent significant advancements in the evaluation of cervical lesions, each offering unique strengths in the diagnosis and management of cervical abnormalities. The MRI, with its refined focus on color, margin, surface features, and vascular patterns, provides a valuable framework for assessing lesions. Meanwhile, the MSCI enhances this approach by incorporating lesion size and location, leading to improved diagnostic accuracy and practicality in clinical settings. Together, these indices not only contribute to the standardization of colposcopic assessments but also play a critical role in the broader effort to enhance cervical cancer screening and treatment strategies. Their adoption in clinical practice is likely to have a profound impact on reducing the incidence of cervical cancer and improving patient outcomes through timely and accurate diagnosis.

Aim of the Study

To assess diagnostic accuracy of Modified Swede Score & Modified Reid Index in comparison with Pap Smear, for pre invasive cervical lesions at AIIMS Bhopal.

Objectives of the Study

To calculate the Modified Swede Score in women attending Gynaecology OPD at AIIMS Bhopal having pre- invasive cervical lesions.

To calculate the Modified Reid Index in women attending Gynaecology OPD at AIIMS Bhopal having pre- invasive cervical lesions.

To compare the Modified Swede Score and Modified Reid Index in these women to assess the diagnostic accuracy of Modified Swede Score & Modified Reid Index, when assuming cytological examination (Pap Smear) as the gold standard.

Methods

Study Design

This study was a hospital-based prospective cross-sectional study. Data was collected from participants who met the inclusion criteria over a defined period. Observations and outcomes were systematically recorded and analyzed to address the study objectives.

Study Participants

A total of 300 sexually active women, aged between 25 to 60 years, were recruited for the study. Participants were selected based on the defined inclusion and exclusion criteria.

Inclusion Criteria

Participants met the following criteria to be included in the study:

1. Participants aged 25 to 60 years who are sexually active.

2. Participants with abnormal Pap smear results (ASCUS or higher).

3. Participants with no gross lesions observed on the cervix and vagina and no history of post-coital bleeding.

4. Participants who exhibit a healthy cervix with no signs suspicious of malignancy upon naked eye examination.

Exclusion Criteria

Participants were excluded from the study if they met any of the following conditions:

1. Participants experiencing ongoing vaginal bleeding.

2. Participants who have undergone a gynecological examination within the last week.

3. Participants presenting with any obvious cervical or vaginal growth.

4. Participants who have had previous procedures on the cervix, such as excision biopsy, cryotherapy, or conisation.

5. Participants who test positive for HPV DNA.

6. Participants showing acetowhite areas on the cervix after the application of acetic acid.

7. Participants who are unwilling to participate in the study.

8. Participants who refuse clinical evaluation, including laboratory tests, as part of the management protocol.

Modified Swede Score (Bowring *et al.*, 2010; Kudela *et al.*, 2020)

Parameters	Score		
	0	1	2
Uptake of acetic acid	None/transparent	Shady, milky (not transparent, not opaque)	Distinct, opaque white
Margins and surface on VIA	Diffuse	Sharp but irregular, jagged, "geographical" satellites	Sharp and even; difference in surface level, including "cuffing"
Vessels on VIA	Fine/Regular	Absent	Coarse/Atypical
Lesion size on VIA	< 5 mm	5-15 mm or spanning upto 2 quadrants	> 15 mm or spanning 3-4 quadrants, or undefined endocervically
Iodine staining on VILI	Brown	Faint/Patchy Yellow	Distinct Yellow

Interpretation-

Score 0-4: Low likelihood of high-grade disease. These findings are typically associated with benign or low-grade squamous intraepithelial lesions (LSIL). Routine follow-up or HPV testing may be advised, but biopsy is often not immediately necessary.

Score 5-6: Moderate risk of high-grade disease. These cases may represent intermediate lesions such as low- to high-grade squamous intraepithelial lesions (CIN1 or CIN2). A colposcopy-directed biopsy is recommended for further evaluation.

Score 7-10: High likelihood of high-grade intraepithelial neoplasia (CIN2+), including high-grade squamous intraepithelial lesions (HSIL) or invasive cancer. Immediate colposcopy-directed biopsy and potentially more aggressive intervention (such as excisional procedures) are indicated.

Modified Reid Coloposcopic Index (Kudela *et al.*, 2020)

Parameters	Score		
	0	1	2
Margins on VIA	Condylomatous or micropapillary contour. Flocculated or feathered, jagged, angular, satellite lesion	Regular lesion with smooth indistinct borders	Rolled, peeling edges, sharp margins
Colour on VIA	Shiny, snow white, areas of faint (semi-transparent) whitening	Intermediate shade (shiny but gray white)	Dull, oyster gray
Vessels on VIA	Uniform, fine caliber non-dilated capillary loops, fine punctuation or mosaic	Absence of surface vessels	Definite, coarse punctuation or mosaic

Iodine staining on VILI	Mahogany brown; mustard yellow stains by a minor lesion (by first three criteria)	Partial iodine uptake (mottled pattern)	Mustard yellow staining of significant lesion (an acetowhite area scoring 3 or more points by first three criteria)

Interpretation-

Score 0-2: Likely benign or low-grade squamous intraepithelial lesion (LSIL). Follow-up or HPV testing may be recommended, but immediate intervention is often unnecessary.

Score 3-4: Indicative of intermediate risk. The lesion could be a low- to moderate-grade squamous intraepithelial lesion. Additional diagnostic measures, such as biopsy, are often recommended.

Score 5-8: Suggests a high-grade squamous intraepithelial lesion (HSIL) or invasive carcinoma. This requires immediate attention and more aggressive diagnostic and treatment procedures, including colposcopy-directed biopsy.

Human Tissue Requirements

Cervical cytology samples were collected as part of routine screening procedures. The samples were used for further analysis related to the study's objectives.

Cervical Sample(s) Collection

1. Patient Preparation:

-The patient was positioned in the dorsal lithotomy position, lying on her back with legs flexed and supported by stirrups.

-This positioning ensured optimal access to the cervix for the clinician and contributes to patient comfort during the examination.

2. Sample Collection from the Ectocervix:

-Ayre’s spatula, typically made of plastic or wood, was used to scrape cells from the ectocervical surface.

-The spatula was rotated 360 degrees around the cervix to ensure comprehensive sampling from the transformation zone.

This area was critical due to its susceptibility to dysplastic changes and HPV infection, and the sample primarily consists of squamous epithelial cells.

3. Sample Collection from the Endocervix:

-A cytobrush, designed with soft bristles, was introduced into the cervical os and gently rotated to collect cells from the endocervical canal.

-This sampling captured columnar epithelial cells, which can reveal HPV-related changes not present in the ectocervical sample alone.

4. Specimen Handling:

-There are two primary methods for specimen handling: conventional Pap smear and liquid-based cytology.

-In the conventional method, collected cells were smeared onto a glass slide and fixed with a solution to preserve morphology.

-The liquid-based cytology method involved rinsing the spatula and cytobrush in a preservative solution, dispersing cells into a liquid medium, which reduced contamination and improved sample quality.

5. Staining and Analysis:

-Samples underwent preparation steps, including fixation, hydration, and staining with the Papanicolaou stain.

-The staining process included fixation, hematoxylin staining for nuclear detail, and counterstaining with orange G-6 and EA-36 for cytoplasmic visualization.

-Slides were examined under a microscope by a pathologist, assessing cell morphology for signs of dysplasia, koilocytosis, or malignant features.

6. Interpretation and Reporting:

-Results were classified using the Bethesda System for Reporting Cervical Cytology.

-Categories include negative for intraepithelial lesion or malignancy (NILM), atypical squamous cells of undetermined significance (ASCUS), low-grade squamous intraepithelial lesion (LSIL), high-grade squamous intraepithelial lesion (HSIL), and squamous cell carcinoma (SCC).

Image 1- Normal ectocervix- normal superficial eosinophilic cells. Some debris in the background. (obj. 20x). (WHO, n.d.)

Image 2- LSIL- eosinophilic squamous cells with a perinuclear empty cavity surrounded by cytoplasmic thickening and with moderate nuclear enlargement- typical koilocytes. (obj. 20x). (WHO, n.d.)

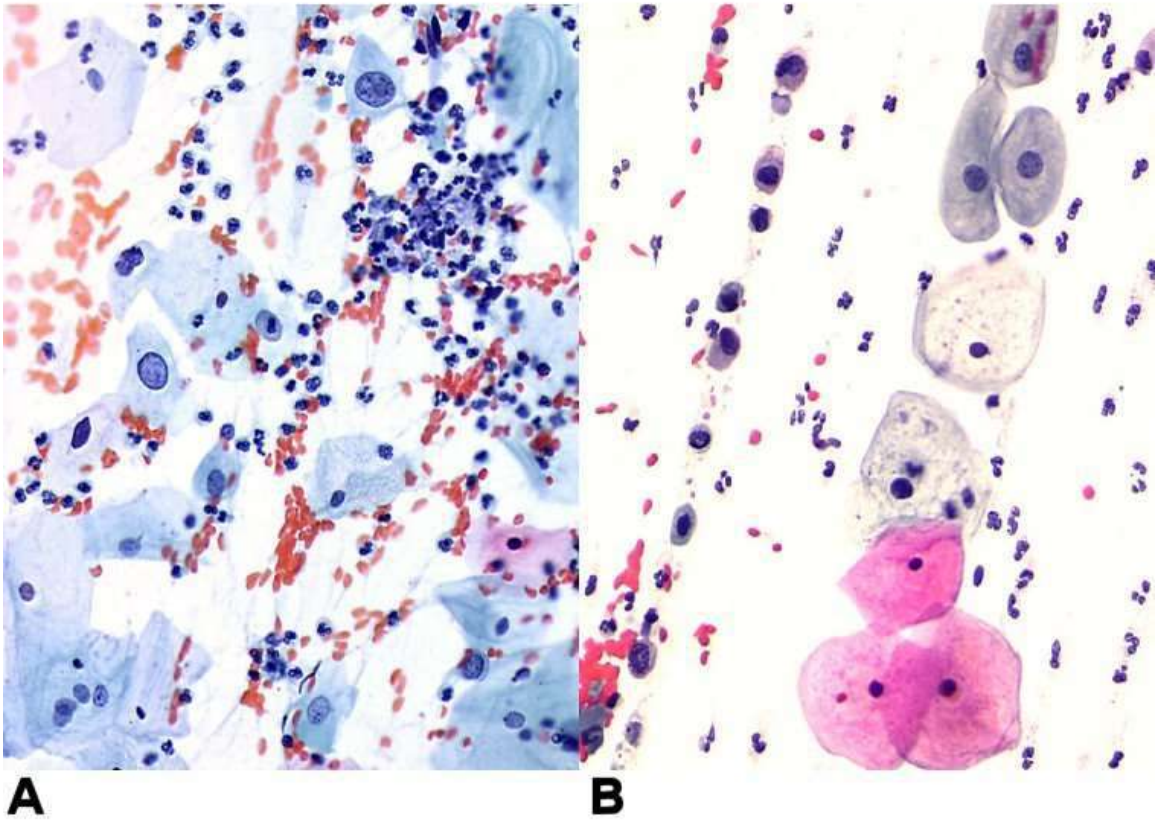

Image 3- ASCUS- Atypical squamous cells of undetermined significance. (obj. 20x). (WHO, n.d.)

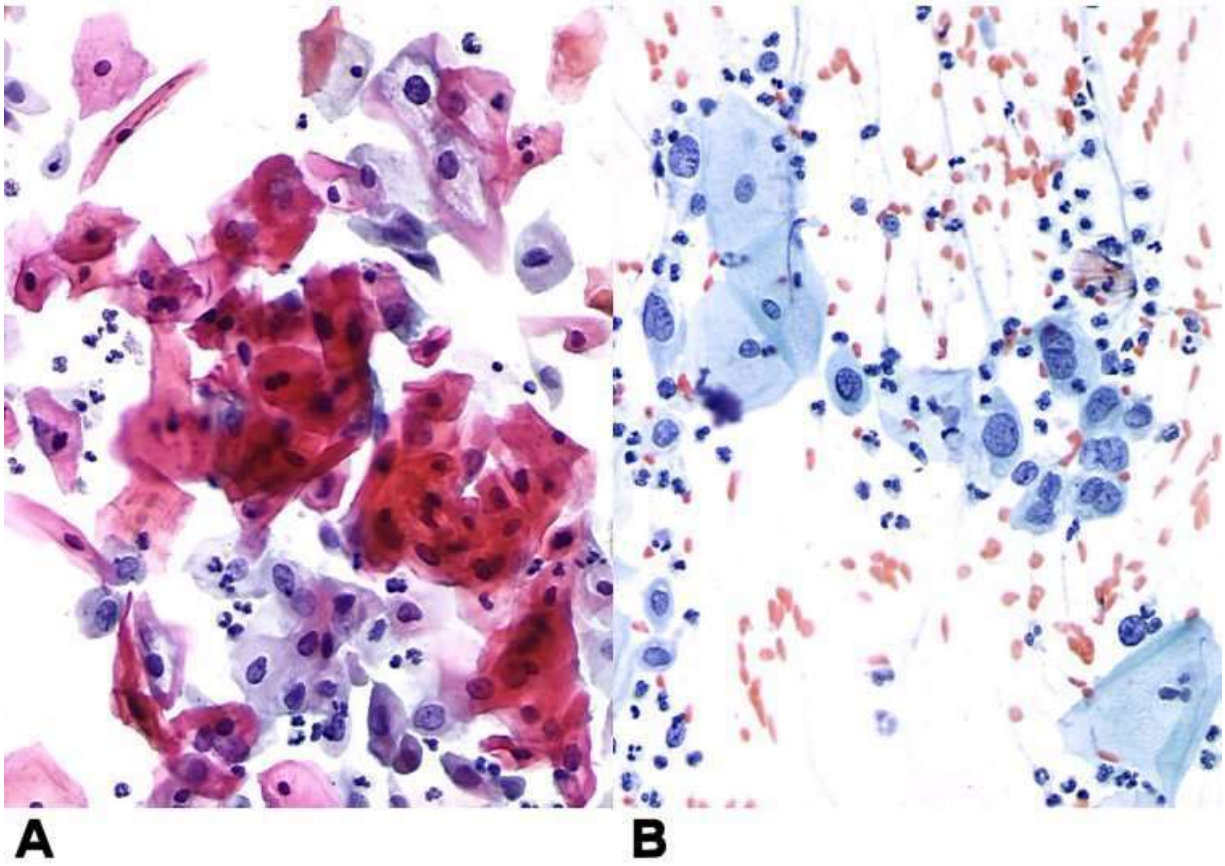

Image 4- A- Low-Grade Squamous Intraepithelial Lesion (LSIL), B- High-Grade Squamous Intraepithelial Lesion (HSIL). (WHO, n.d.)

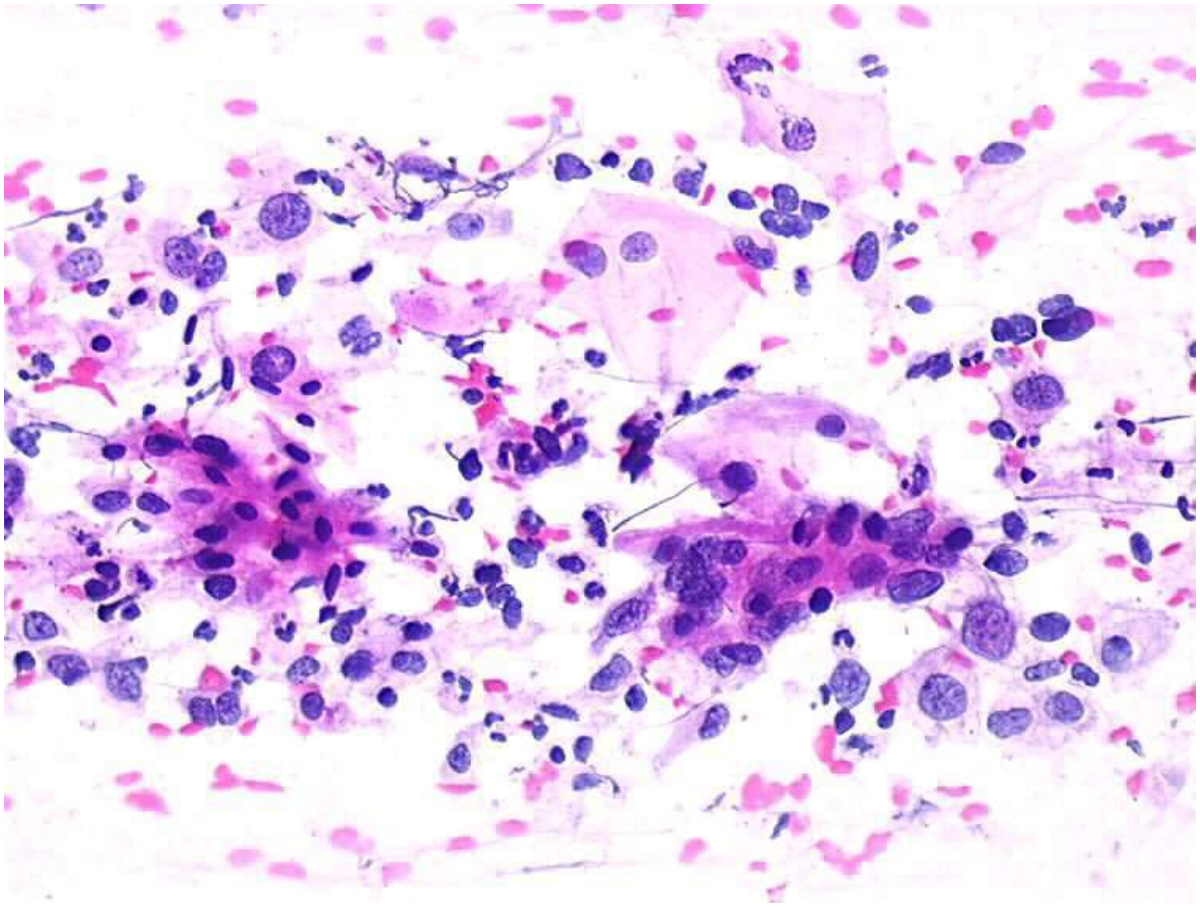

Image 5- Invasive SCC- pleomorphic malignant cells, isolated or in clusters, sometimes with markedly atypical nuclei or elongated nuclei. Inflammation, blood and necrosis in the background. (obj. 20x). (WHO, n.d.)

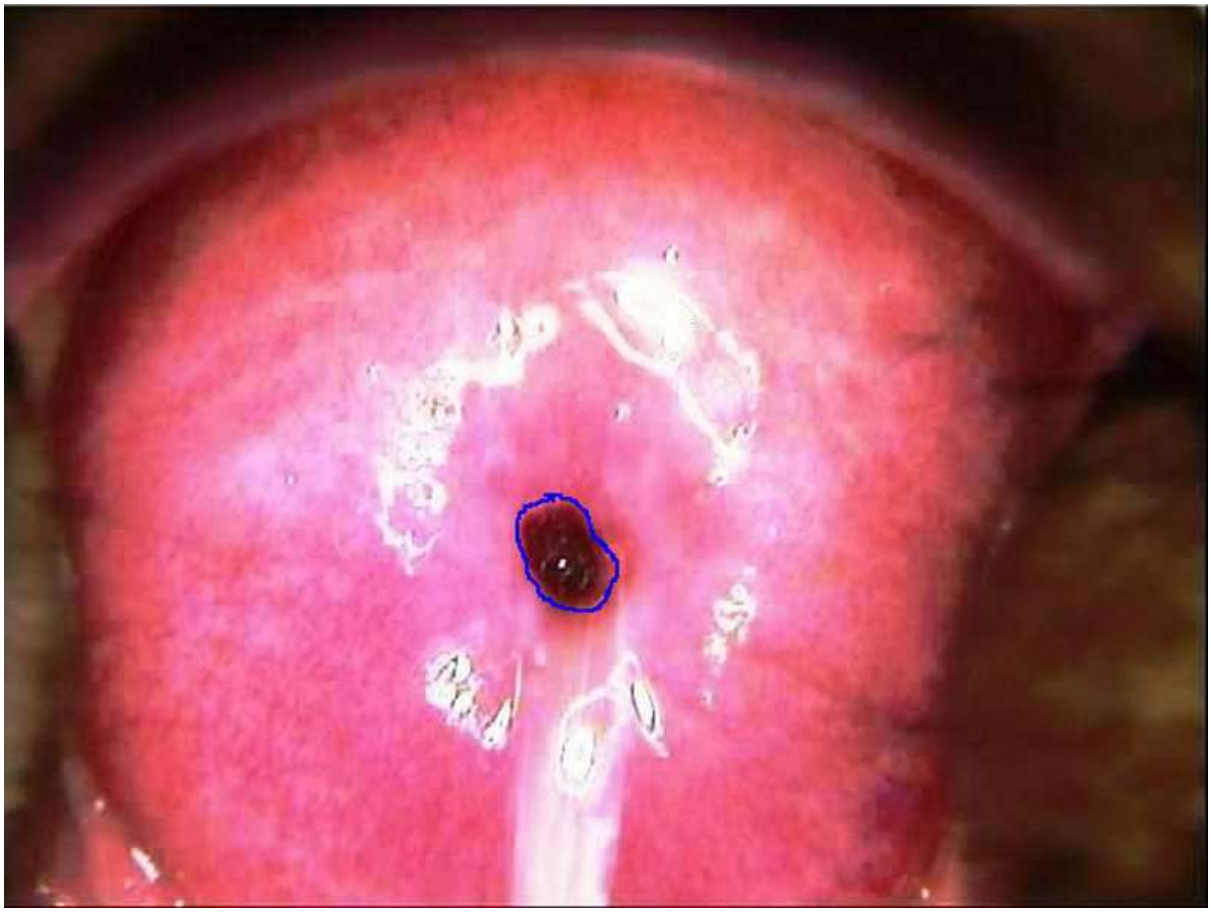

Image 6- Normal Cervix- After Normal Saline. The external os is small and round. The SCJ is visible at the external os. Blue Line- SCJ. (IARC, n.d.)

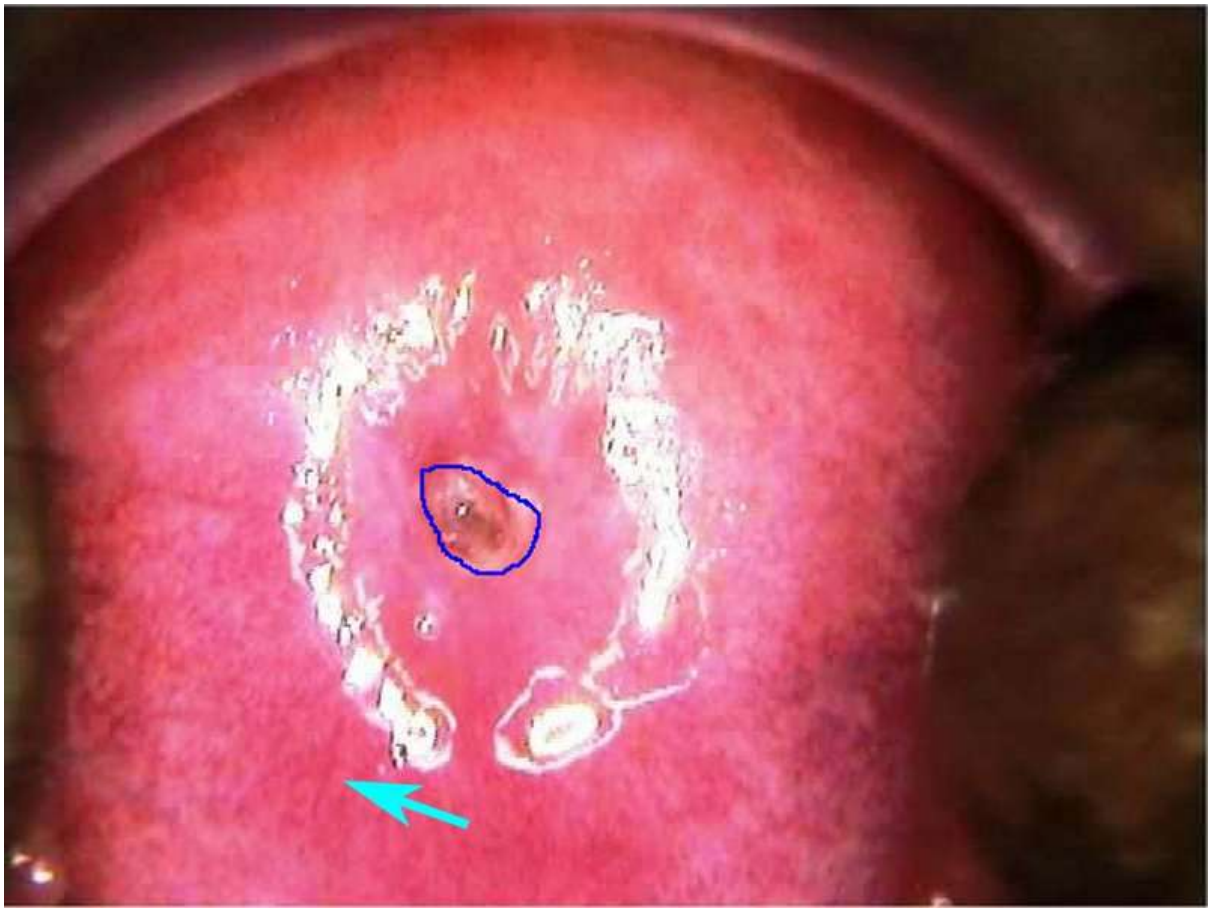

Image 7- Normal Cervix- After Acetic Acid. No acetowhite area. Mature squamous epithelium with normal capillaries is seen. Blue Line- SCJ. Blue arrow- Normal capillaries. (IARC, n.d.)

Image 8- Normal Cervix- After Lugol's Iodine. The squamous epithelium has turned brown, and the columnar epithelium has not taken up any stain. (IARC, n.d.)

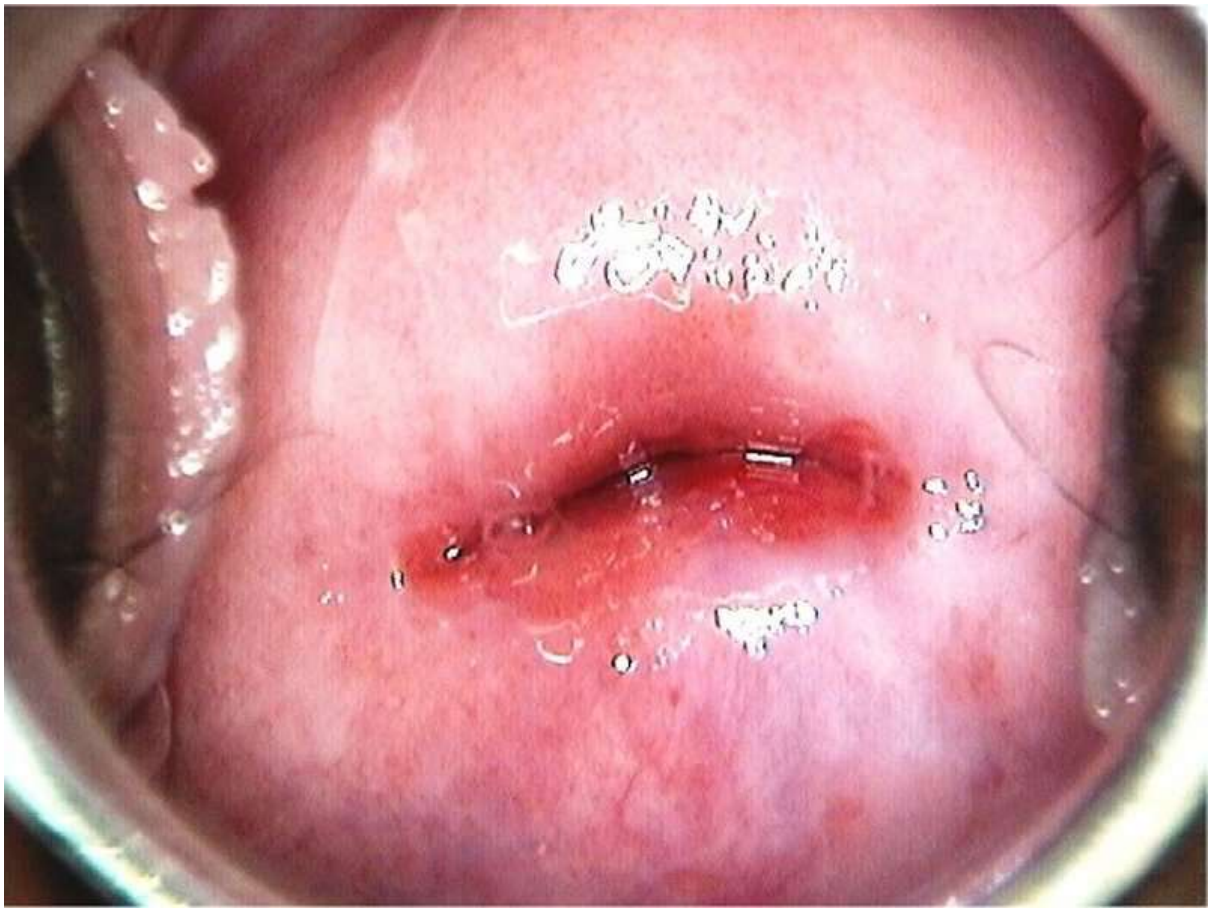

Image 9- LSIL- After Normal Saline. The SCJ is fully visible at the external os. (IARC, n.d.)

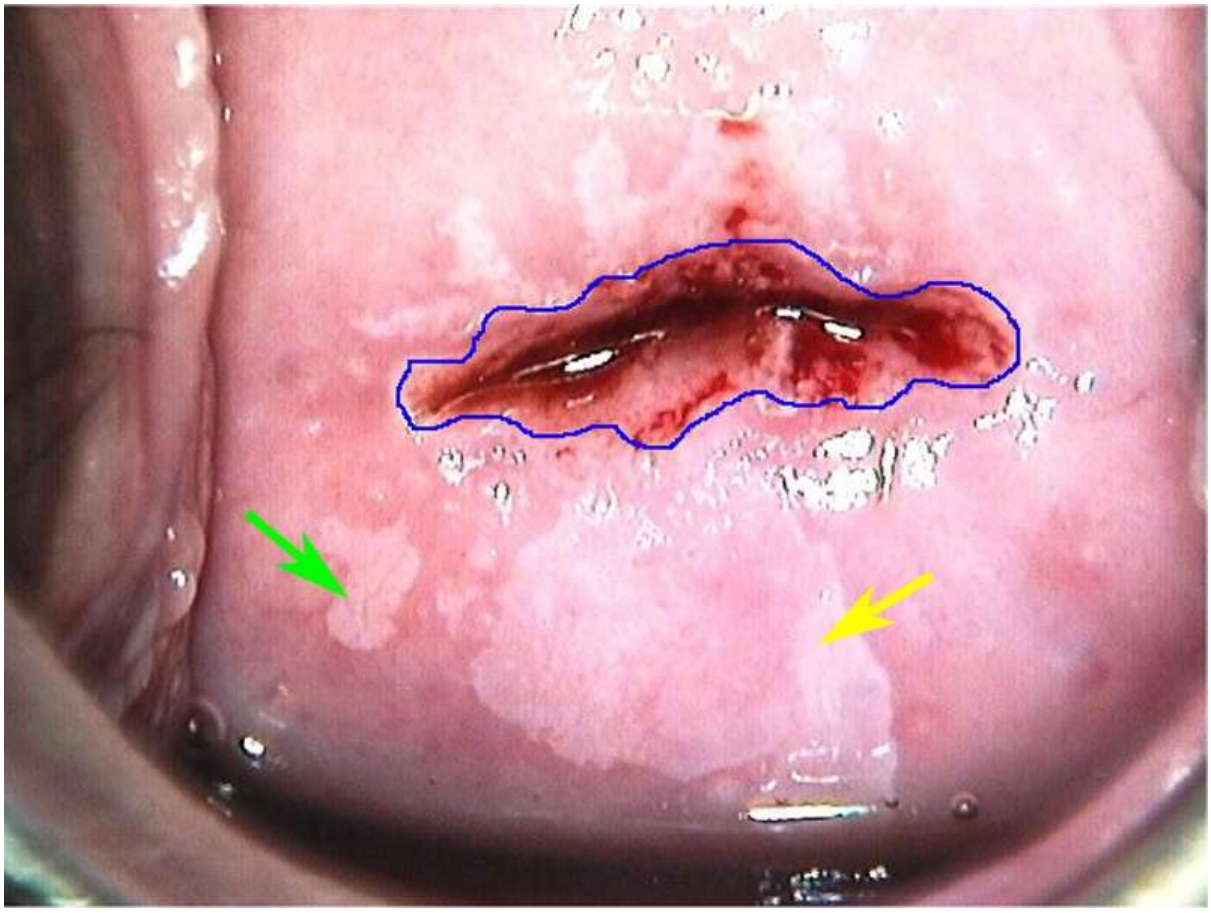

Image 10- LSIL- After Acetic Acid. Thin acetowhite areas are seen at the 6 o'clock and 12 o'clock positions. The lesion on the posterior lip has a sharp but irregular border. The lesion on the anterior lip has an irregular border. Both lesions arise from the SCJ. A satellite lesion is seen at the 8 o'clock position. Blue line: SCJ. Yellow arrow: Acetowhite area. Green arrow: Satellite. (IARC, n.d.)

Image 11- LSIL- After Lugol's Iodine. There is patchy iodine uptake on the lesions. (IARC, n.d.)

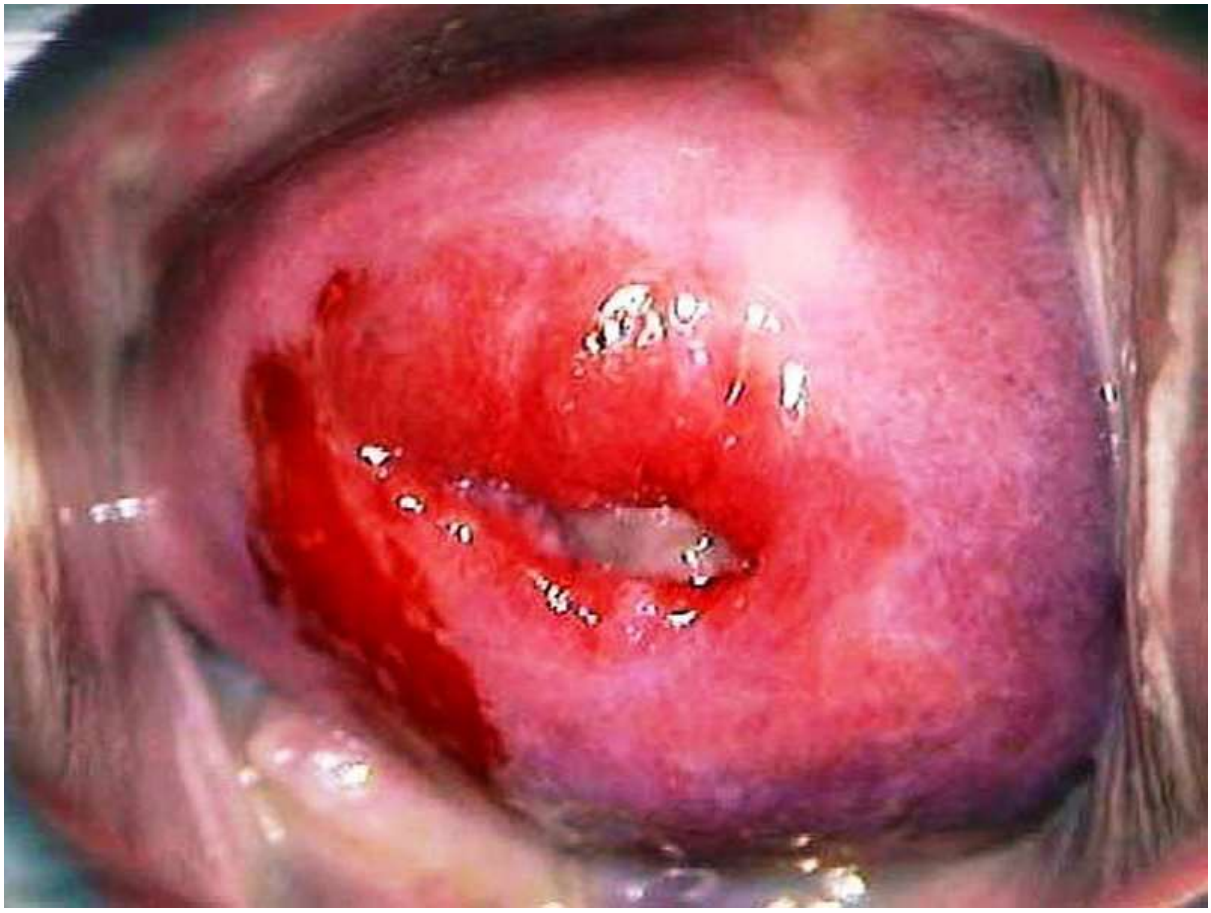

Image 12- HSIL- After Normal Saline. The central red area on the cervix is not always due to columnar epithelium. Metaplasia or even CIN lesions can appear as a red patch on the cervix. In this picture the SCJ seems to be on the ectocervix. (IARC, n.d.)

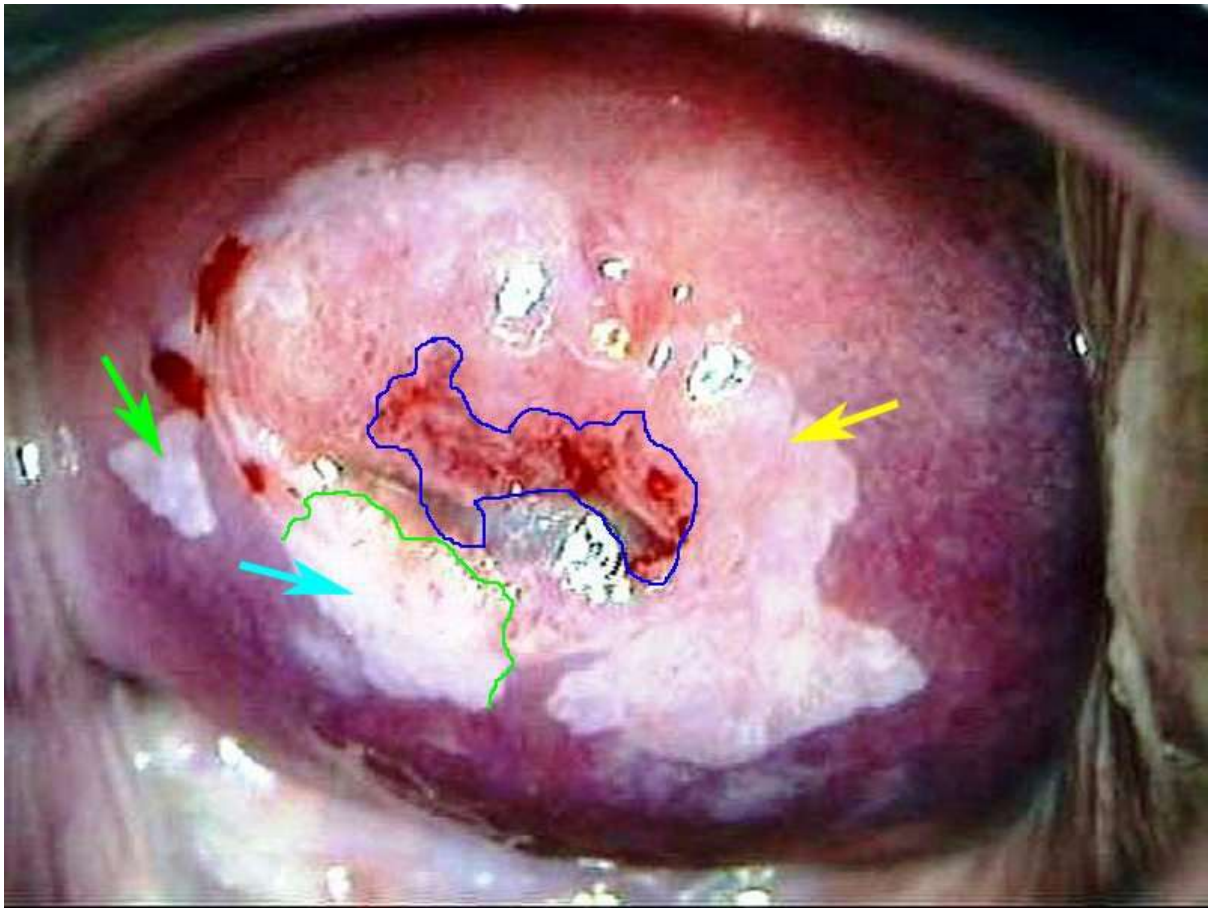

Image 13- HSIL- After acetic acid. There is a large acetowhite area with a regular margin extending to almost all quadrants. The lesion at the 7 o'clock position is denser than the surrounding areas, resulting in the inner border sign. There is a small acetowhite area at the 9 o'clock position. Blue line: SCJ. Green line: Inner border sign. Yellow arrow: Thin acetowhite area. Blue arrow: Dense acetowhite area. Green arrow: Acetowhite area with geographical margin away from the transformation zone. (IARC, n.d.)

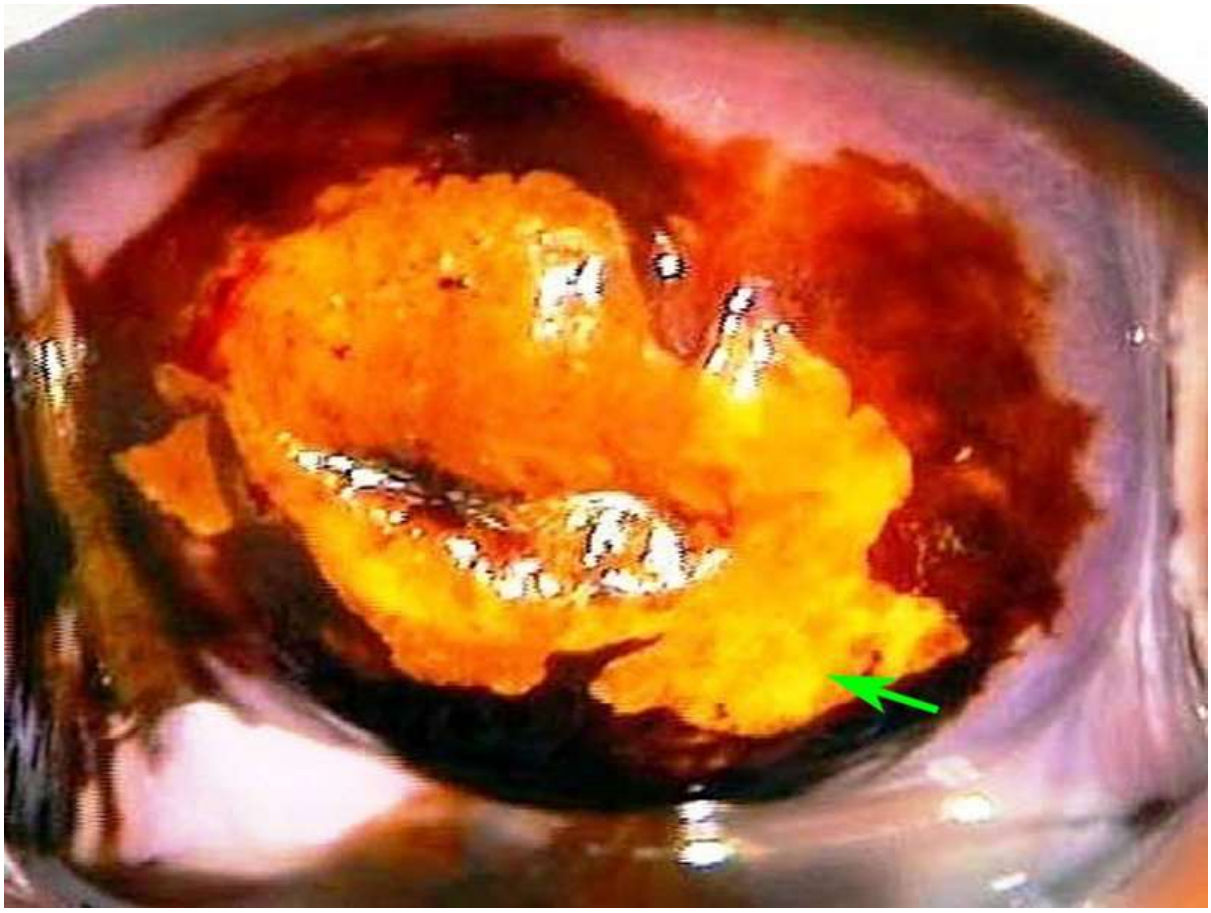

Image 14- HSIL- After Lugol's Iodine. The acetowhite areas have not been stained with Lugol's iodine. Green arrow: Iodine-negative. (IARC, n.d.)

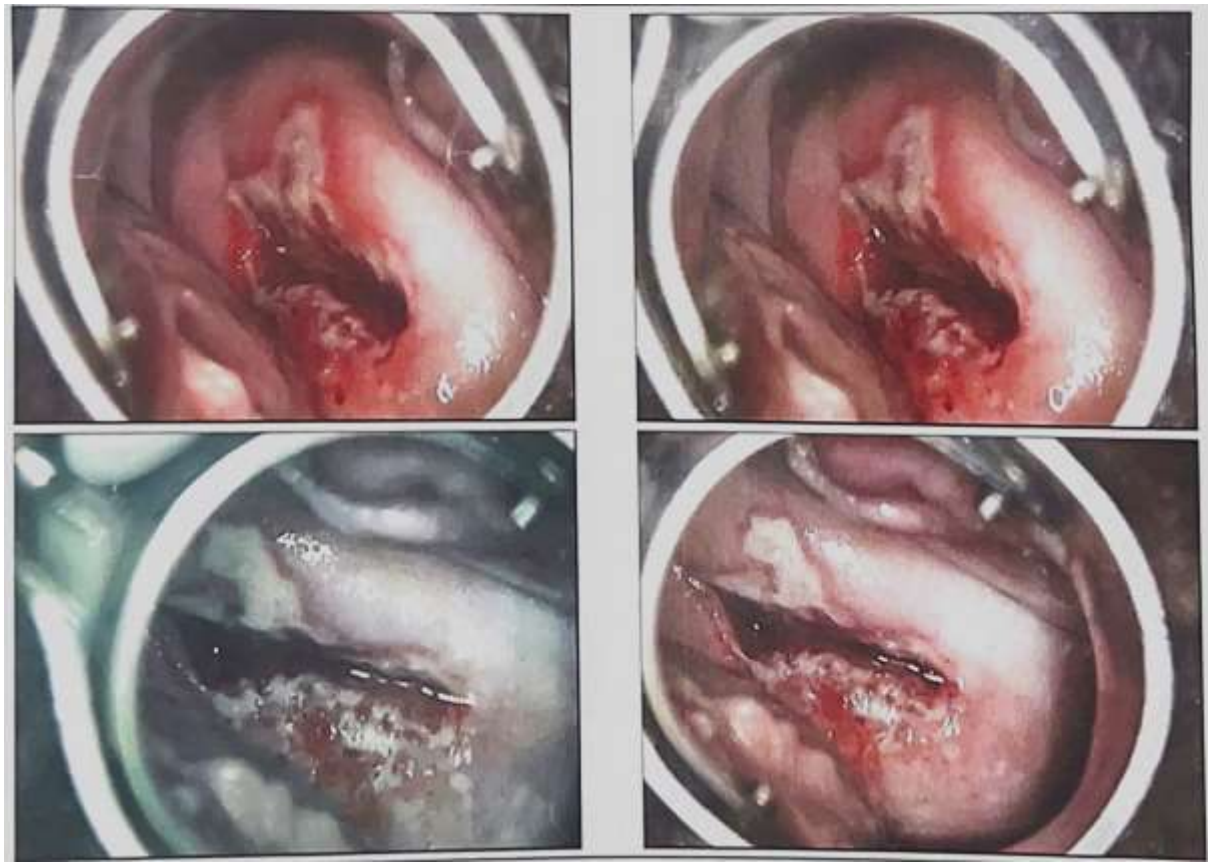

Image 15- HSIL- Colposcopy; Pap Smear revealed HSIL.

Image 16- LSIL- Colposcopy; Pap Smear revealed LSIL

Data Collection & Analysis

Data collection was carried out with an individual questionnaire asking details about sexual history, number of partners, past medical/surgical history. Final details were entered into an Excel spreadsheet. Measures to maintain the anonymity of the data collected, including de-identification and restricted access to sensitive information were implemented. However, it was important to note that complete confidentiality couldn't be guaranteed due to the inherent risks associated with data handling and risk of potential breaches.

Data analysis were conducted using IBM SPSS or comparable statistical software to thoroughly evaluate key diagnostic metrics, including sensitivity, specificity, accuracy, positive predictive value, negative predictive value. Additionally, receiver operating characteristic (ROC) analysis were performed as needed to assess the performance characteristics of the diagnostic tests, enabling us to determine optimal threshold values and evaluate the trade-offs between sensitivity and specificity.

Results

Descriptives

Descriptives

	Pap Smear	N	Missing	Mean	Median	SD	Minimum	Maximum
AGE	Negative	232	2	44.9	45.0	9.53	22	85
	Positive	66	0	44.3	44.5	8.71	25	63

Table 1- Descriptive Statistics of Age by Pap Smear Results

A total of 300 participants were included in this analysis, with 232 individuals receiving negative Pap smear results and 66 individuals receiving positive results. Additionally, there were 2 cases of missing data for the negative group and no missing cases for the positive group.

Age Analysis

The mean age of participants with negative Pap smear results was 44.9 years (SD = 9.53), while the mean age for those with positive results was slightly lower at 44.3 years (SD = 8.71). The median age for the negative group was 45.0 years, compared to 44.5 years for the positive group, indicating a relatively symmetrical distribution of ages within both groups.

The age range for the negative group was from 22 to 85 years, while the positive group had an age range of 25 to 63 years. These findings suggest that the negative group included younger individuals, while the positive group had a more limited age range with older participants.

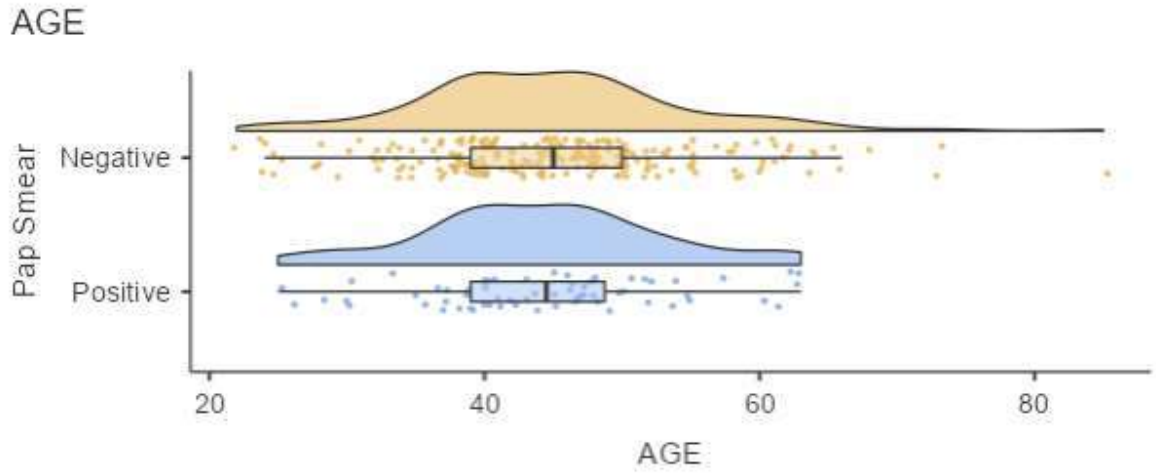

Fig 1- Box & Whisker Plot of Age Distribution in Pap Smear Results

Frequencies

Frequencies of Parity (Nullipara vs Unipara vs Multipara)

Parity (Nullipara vs Unipara vs Multipara)	Pap Smear	Counts	% of Total	Cumulative %
Nullipara	Negative	6	2.0 %	2.0 %
	Positive	5	1.7 %	3.7 %
Unipara	Negative	16	5.3 %	9.0 %
	Positive	1	0.3 %	9.3 %
Multipara	Negative	212	70.7 %	80.0 %
	Positive	60	20.0 %	100.0 %

Table 2- Frequencies of Parity and Pap Smear Results

The distribution of participants by parity (Nullipara, Unipara, and Multipara) in relation to their Pap smear results (negative or positive) is presented.

Among the participants, 6 Nullipara individuals (2.0% of the total) had negative Pap smear results, while 5 (1.7% of the total) tested positive, accounting for a cumulative percentage of 3.7%. This indicates that Nullipara individuals represent a relatively small portion of the overall population, with a slightly higher prevalence of negative results compared to positive.

In the Unipara category, 16 participants (5.3% of the total) received negative Pap smear results, and only 1 participant (0.3% of the total) tested positive, bringing the cumulative percentage to 9.3%. This suggests that Unipara individuals also constitute a small portion of the sample, with the vast majority obtaining negative Pap smear results.

The Multipara group included 212 individuals (70.7% of the total) with negative Pap smear results, while 60 participants (20.0% of the total) tested positive, resulting in a cumulative percentage of 100.0%. This finding indicates that Multipara individuals are the largest group within the study, with a significant majority of negative results and a notable proportion of positive results.

The data reveals a clear predominance of Multipara individuals among the study population, particularly within the negative Pap smear results category. Conversely, Nullipara and Unipara participants are less represented, especially among those with positive results. This distribution underscores the potential implications of parity in cervical health and screening outcomes. Given the significant representation of Multipara individuals, future research should further investigate the relationship between parity and the incidence of abnormal Pap smear results, as well as the underlying factors contributing to these outcomes.

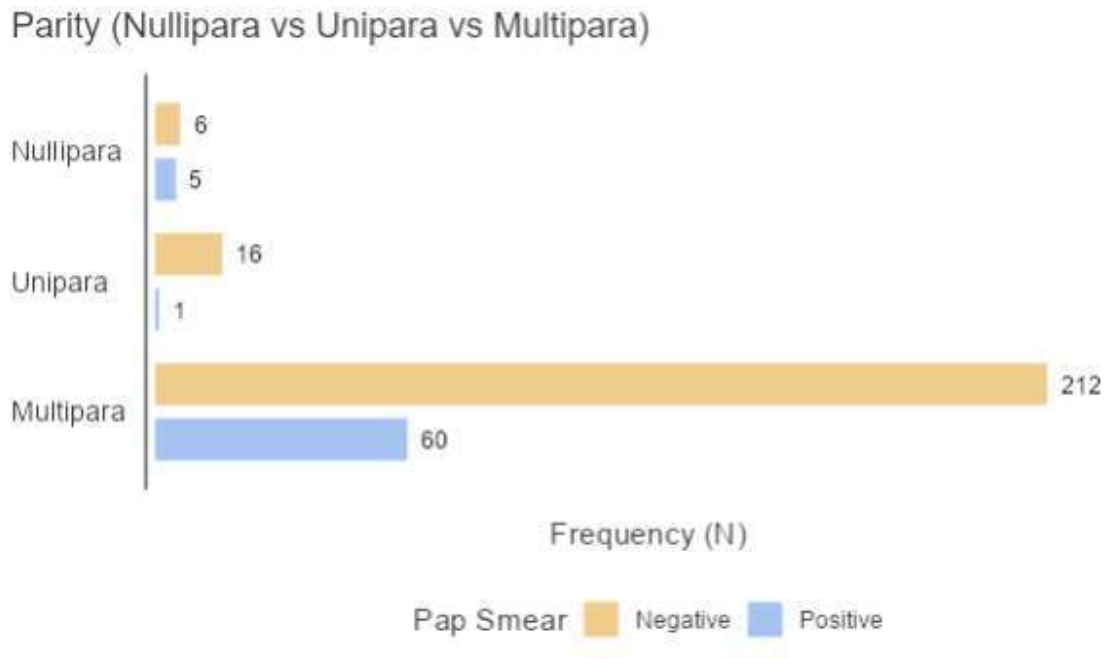

Fig 2- Bar Plot of Parity Distribution by Pap Smear Results

Frequencies of USE OF CONTRACEPTION

USE OF CONTRACEPTION	Pap Smear	Counts	% of Total	Cumulative %
No	Negative	212	70.7 %	70.7 %
	Positive	65	21.7 %	92.3 %
Yes	Negative	22	7.3 %	99.7 %
	Positive	1	0.3 %	100.0 %

Frequencies of USE OF CONTRACEPTION				
USE OF CONTRACEPTION	**Pap Smear**	**Counts**	**% of Total**	**Cumulative %**
Table 3- Frequencies of Contraception Use and Pap Smear Results				

Among the participants who do not use contraception, 212 individuals (70.7% of the total) had negative Pap smear results, while 65 individuals (21.7% of the total) tested positive. This accounts for a cumulative percentage of 92.3%. This indicates that the majority of participants who do not use contraception have negative Pap smear results, but there is a notable proportion that tested positive.

In contrast, among those who use contraception, 22 participants (7.3% of the total) received negative Pap smear results, and only 1 participant (0.3% of the total) tested positive, leading to a cumulative percentage of 100.0%. This suggests that individuals who use contraception are significantly underrepresented in this sample, particularly among those with positive Pap smear results.

Overall, the findings indicate a predominance of participants who do not use contraception, especially in the negative Pap smear category. The low percentage of positive results among those using contraception suggests a potential protective factor, although the small sample size of contraceptive users limits the ability to draw strong conclusions.

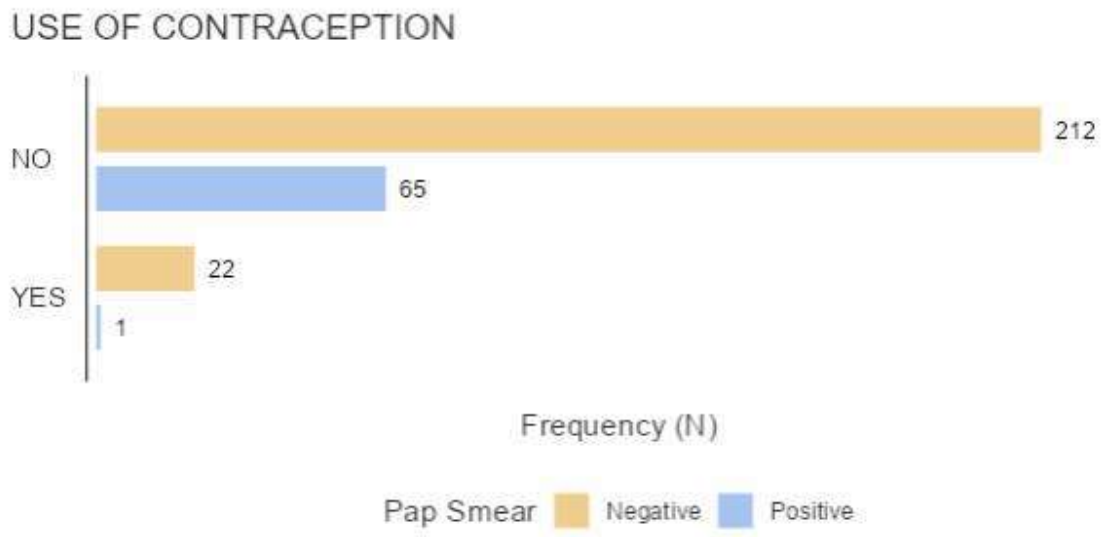

Fig 3- Bar Plot of Distribution of Use of Contraception by Pap Smear Results

Frequencies of ANY H/O SEXUALLY TRANSMITTED DISEASE											
ANY H/O SEXUALLY TRANSMITTED DISEASE		**Pap Smear**		**Counts**		**% of Total**		**Cumulative %**			
No		Negative		234		78.0 %		78.0 %			
		Positive		66		22.0 %		100.0 %			
Table 4- Frequencies of History of Sexually Transmitted Diseases and Pap Smear Results											

100% of the participants (n=300) did not have a prior history a sexually transmitted disease. Of these, 78% (n=234) had a negative Pap smear and rest 22% (n=66) had a positive Pap smear.

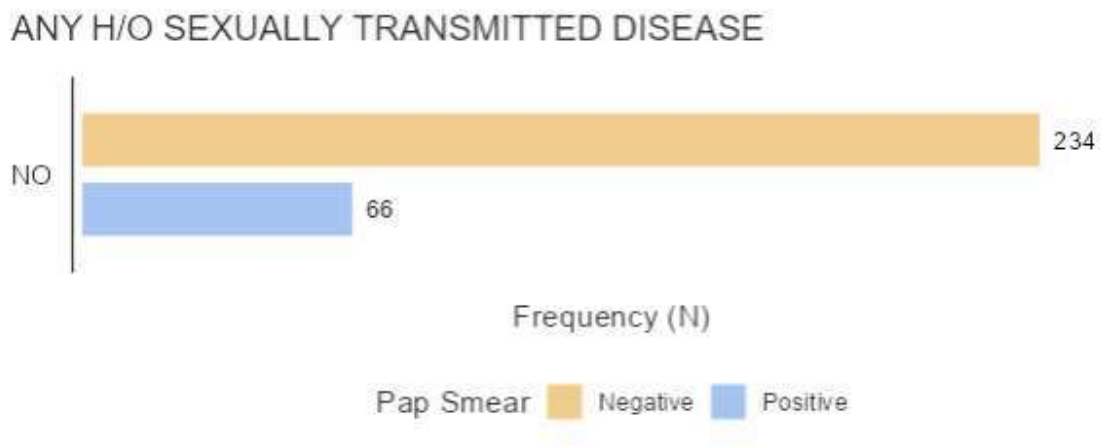

Fig 4- Bar Plot of Distribution of history of STDs by Pap Smear Results

Frequencies of FAMILY HISTORY OF CERVICAL CANCER

FAMILY HISTORY OF CERVICAL CANCER	Pap Smear	Counts	% of Total	Cumulative %
NO	Negative	234	78.0 %	78.0 %
	Positive	66	22.0 %	100.0 %

Table 5- Frequencies of Family History of Cervical Cancer and Pap Smear Results

100% of the participants (n=300) did not have a family history of cervical cancer. Of these, 78% (n=234) had a negative Pap smear and rest 22% (n=66) had a positive Pap smear.

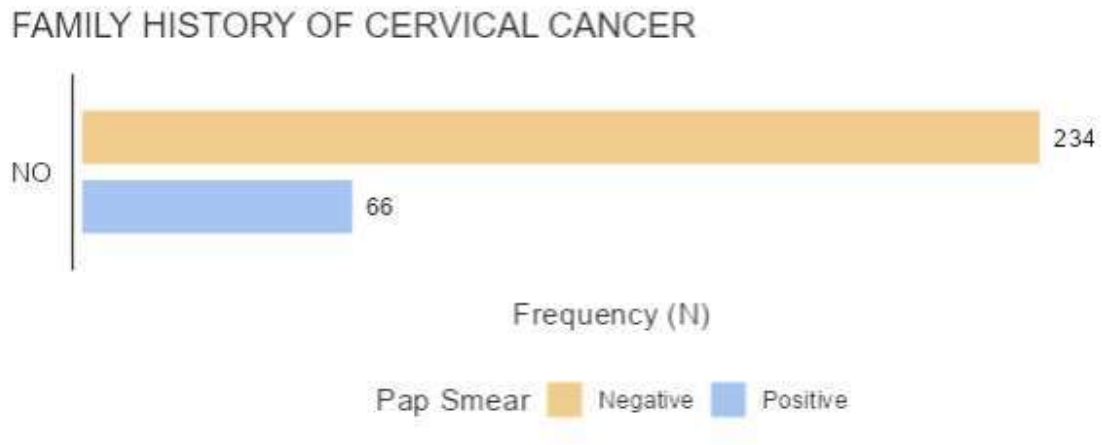

Fig 5- Bar Plot of Distribution of history of Cervical Cancer by Pap Smear Results.

Frequencies of PAP SMEAR

PAP SMEAR	Counts	% of Total	Cumulative %
ASCUS	1	0.3 %	0.3 %

Frequencies of PAP SMEAR			
PAP SMEAR	**Counts**	**% of Total**	**Cumulative %**
HSIL	14	4.7 %	5.0 %
LSIL	49	16.3 %	21.3 %
LSIL + BACTERIAL VAGINOSIS	2	0.7 %	22.0 %
NEGATIVE	234	78.0 %	100.0 %

Table 6- Frequencies of Pap Smear Results

The data presents the frequencies of various Pap smear results in relation to the total sample of participants. Among the results, 1 participant (0.3% of the total) received an ASCUS (Atypical Squamous Cells of Undetermined Significance) result, indicating a very small proportion of cases. For HSIL (High-Grade Squamous Intraepithelial Lesion), 14 participants (4.7% of the total) had this result, reflecting a higher level of concern for potential cervical cancer. In the case of LSIL (Low-Grade Squamous Intraepithelial Lesion), 49 participants (16.3% of the total) tested positive, representing a moderate proportion of cases that may require monitoring. Additionally, 2 participants (0.7% of the total) were noted to have both LSIL and bacterial vaginosis, indicating a combination of findings. The majority, 234 participants (78.0% of the total), received negative Pap smear results, suggesting no significant abnormalities. The cumulative percentage shows that 78.0% of the participants had negative results, while a total of 22.0% were identified with varying degrees of abnormalities (ASCUS, LSIL, HSIL, and combined LSIL with bacterial vaginosis).

Overall, the findings indicate that a substantial majority of participants have negative Pap smear results, while a smaller proportion exhibit abnormal findings, particularly LSIL and HSIL.

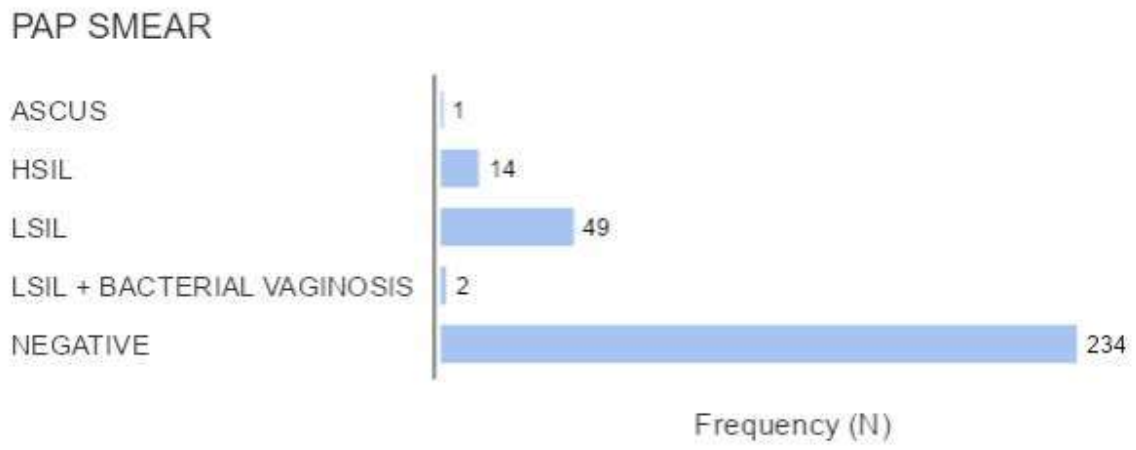

Fig 6- Bar Plot of Distribution of Pap Smear Results

Scale: REID'S INDEX							
Cutpoint	**Sensitivity (%)**	**Specificity (%)**	**PPV (%)**	**NPV (%)**	**Youden's index**	**AUC**	**Metric Score**
1	100%	0%	22%	NaN%	0.00000	0.993	1.00
2	100%	0.85%	22.15%	100%	0.00855	0.993	1.01
3	100%	7.69%	23.4%	100%	0.07692	0.993	1.08
4	100%	96.15%	88%	100%	0.96154	0.993	1.96
5	81.82%	99.15%	96.43%	95.08%	0.80963	0.993	1.81
6	22.73%	100%	100%	82.11%	0.22727	0.993	1.23
7	13.64%	100%	100%	80.41%	0.13636	0.993	1.14
8	4.55%	100%	100%	78.79%	0.04545	0.993	1.05

Table 7- Scale: Reid's Index Metrics and Performance Indicators

Sensitivity remains high at the lower cutpoints (1-3), indicating that the test is very effective at identifying those with the disease, but specificity is very low at these levels, resulting in many false positives. As cutpoints increase, sensitivity decreases, but specificity improves significantly, especially at cutpoints 4 and 5. PPV improves notably as specificity increases, especially at higher cutpoints, indicating that a positive test result is much more likely to be accurate. NPV is consistently high, particularly at cutpoints 2-4, confirming that negative results are reliable. Youden's Index demonstrates the effectiveness of the test, peaking at cutpoint 4, suggesting it offers the best balance between sensitivity and specificity. The AUC remains high across all cutpoints (0.993), indicating strong overall performance of the Reid's Score as a diagnostic tool.

In conclusion, cutpoint 4 appears to offer the best compromise between sensitivity and specificity, making it the most effective choice for diagnosing the disease based on Reid's Score.

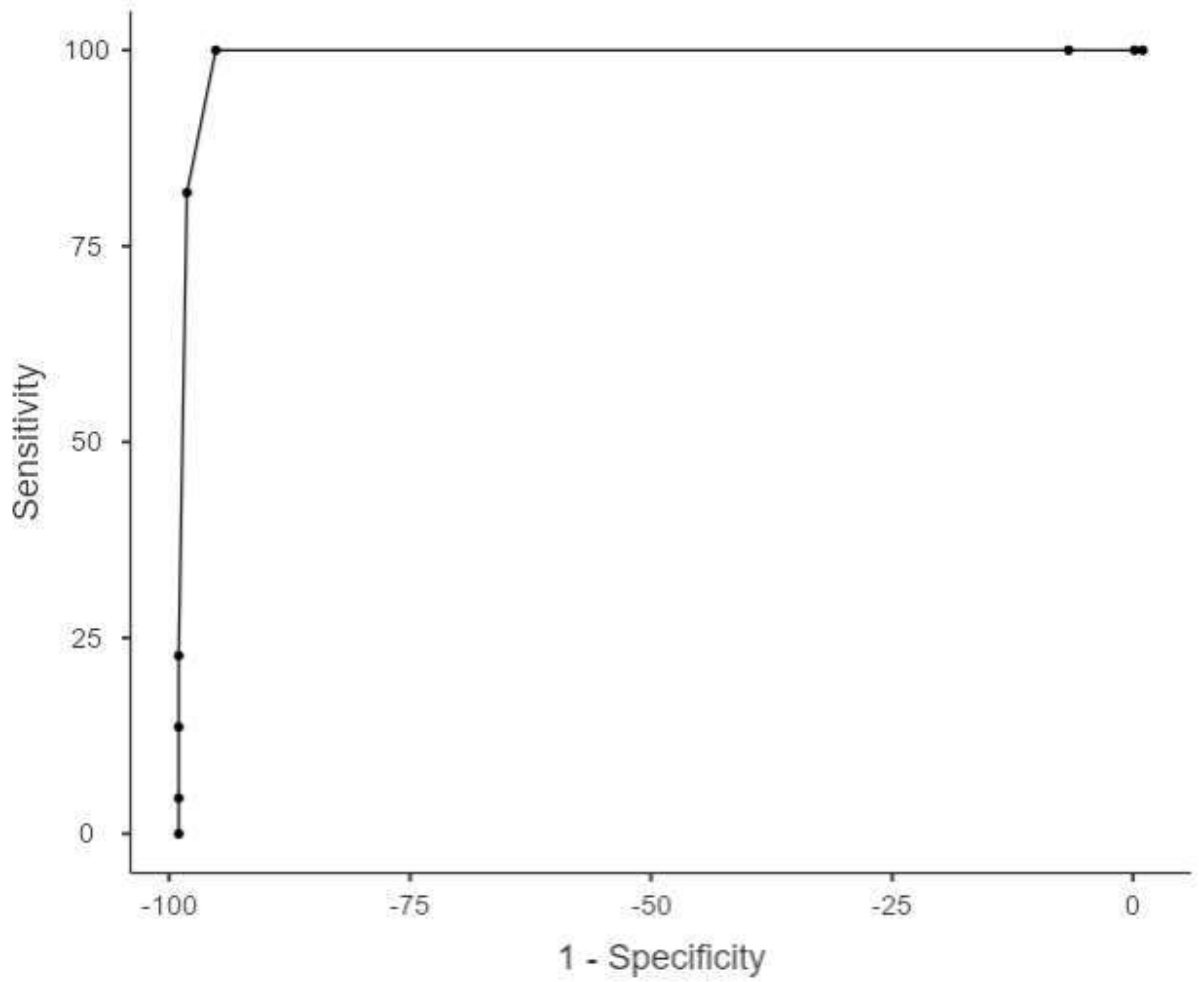

Fig 7- ROC Curve Plot for Evaluating Test Performance: The curve illustrates the trade-off between sensitivity (true positive rate) and 1-specificity (false positive rate) for the Reid's Index, depicting the area under the curve (AUC) as a measure of overall accuracy, providing a visual representation of the test's ability to discriminate between positive and negative cases.

The ROC curve analysis for the Reid's Score reveals an AUC of 0.993, indicating outstanding discrimination between diseased and healthy individuals. The curve approaches the top left corner of the plot, reflecting high sensitivity and specificity. A notable threshold (cut-point 4) for optimal balance occurs at a sensitivity of 100% and a specificity of 96.15%, suggesting this cut-off may be used to maximize true positive rates while controlling false positives. The comparison of ROC curves for this test against an alternative reveals a statistically significant difference ($p < 0.001$), confirming its superior performance.

Scale: SWEDE'S SCORE							
Cutpoint	**Sensitivity (%)**	**Specificity (%)**	**PPV (%)**	**NPV (%)**	**Youden's index**	**AUC**	**Metric Score**
1	100%	0%	22%	NaN%	0.00000	0.998	1.00
2	100%	0.85%	22.15%	100%	0.00855	0.998	1.01

Scale: SWEDE'S SCORE					
Cutpoint	**Sensitivity (%)**	**Specificity (%)**	**PPV (%)**	**NPV (%)**	**Youden's index**	**AUC**	**Metric Score**
3	100%	12.39%	24.35%	100%	0.12393	0.998	1.12
4	100%	51.28%	36.67%	100%	0.51282	0.998	1.51
5	100%	97.01%	90.41%	100%	0.97009	0.998	1.97
6	98.48%	99.15%	97.01%	99.57%	0.97630	0.998	1.98
7	46.97%	100%	100%	86.99%	0.46970	0.998	1.47
8	15.15%	100%	100%	80.69%	0.15152	0.998	1.15
9	4.55%	100%	100%	78.79%	0.04545	0.998	1.05

Table 8- Scale: Swede's Score Metrics and Performance Indicators

Sensitivity is consistently high (100%) at the lower cutpoints (1-5), indicating that the test reliably detects individuals with the disease. However, specificity is very low at these levels, leading to many false positives. As cutpoints increase, sensitivity decreases significantly, especially at cutpoints 6-9, while specificity remains very high. PPV improves significantly at higher cutpoints, particularly at cutpoints 5 and 6, indicating that a positive result is much more likely to be accurate. NPV remains high across cutpoints, particularly at 5 and 6, confirming that negative results are reliable. Youden's Index peaks at cutpoint 5, indicating it offers the best balance between sensitivity and specificity, making it the most effective for disease identification. The AUC remains very high across all cutpoints (0.998), signifying strong overall performance of Swede's Score as a diagnostic tool.

In conclusion, cutpoint 5 is optimal, providing a strong balance between sensitivity and specificity, thus enhancing the reliability of Swede's Score for diagnosing the disease.

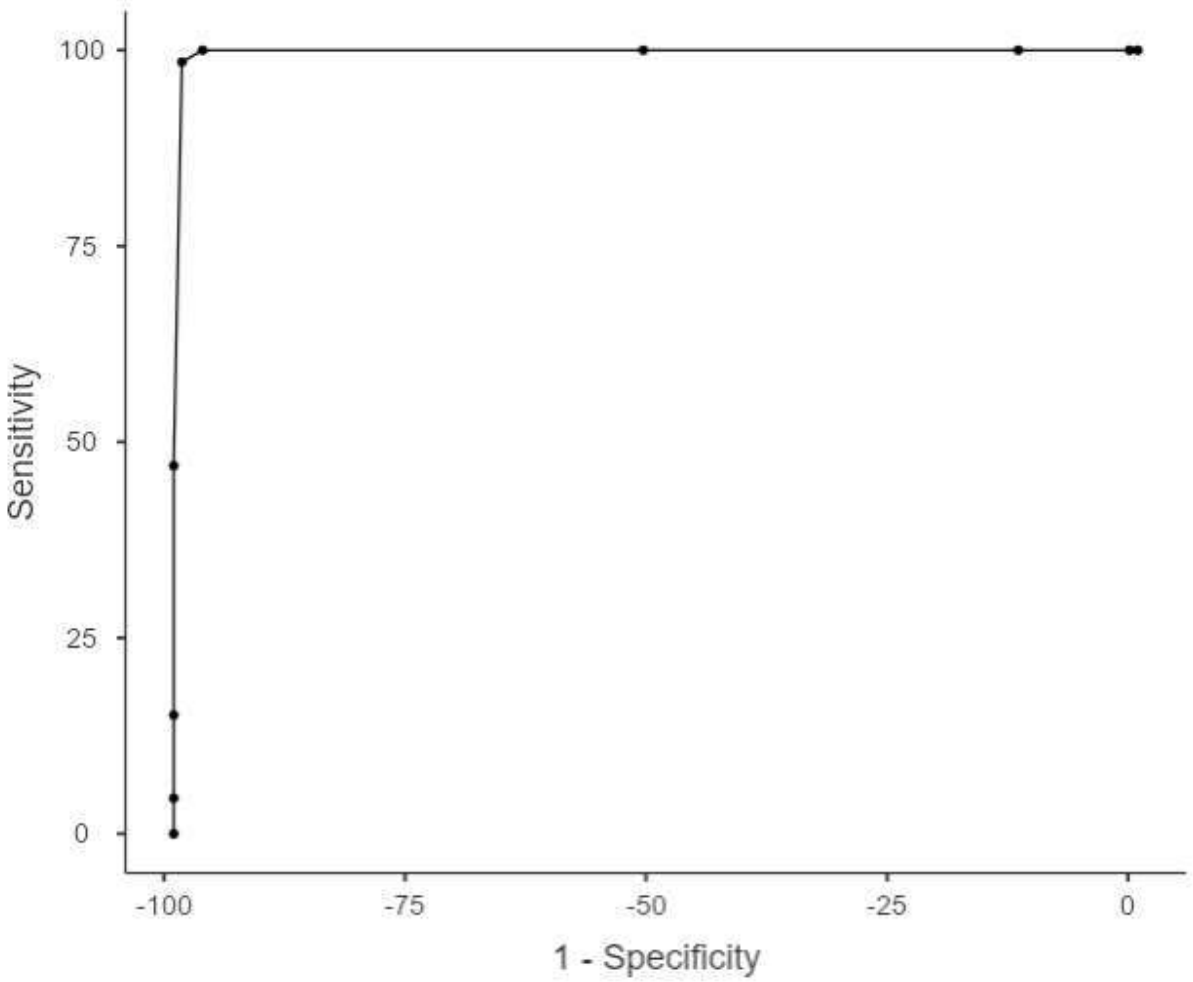

Fig 8- ROC Curve Plot for Evaluating Test Performance: The curve illustrates the trade-off between sensitivity (true positive rate) and 1-specificity (false positive rate) for the Swede's Score, depicting the area under the curve (AUC) as a measure of overall accuracy, providing a visual representation of the test's ability to discriminate between positive and negative cases.

The ROC curve analysis for the Swede's Score reveals an AUC of 0.998, indicating outstanding discrimination between diseased and healthy individuals. The curve approaches the top left corner of the plot, reflecting high sensitivity and specificity. A notable threshold (cut-point 5) for optimal balance occurs at a sensitivity of 100% and a specificity of 97.01%, suggesting this cut-off may be used to maximize true positive rates while controlling false positives. The comparison of ROC curves for this test against an alternative reveals a statistically significant difference ($p < 0.001$), confirming its superior performance.

Estimated AUC's:

	AUC	SD (Hanley)	P(H0: AUC=0.5)	SD(DeLong)	P(H0: AUC=0.5)
1 (Reid's Score)	0.993	0.007	<0.001	0.003	<0.001
2 (Swede's Score)	0.998	0.004	<0.001	0.002	<0.001

Table 9- AUC Comparison for Reid's Index and Swede's Scores with Statistical Analysis

Both Reid's Score and Swede's Score exhibit excellent discriminatory capabilities, with Swede's Score showing a greater performance. The low standard deviations suggest that the AUC estimates are precise, and the statistically significant p-values (<0.001) indicate that both scores meaningfully differentiate between diseased and healthy populations.

Pairwise comparisons:

	AUC Difference	CI (lower)	CI (upper)	P-Value	Correlation
1 vs. 2	-0.005	-0.008	-0.001	0.020	0.893

Table 10- Comparison of AUC Differences Between Reid's Index and Swede's Scores with Statistical Significance

Overall test: p-value = 0.0202

The analysis indicates that Swede's Score has a statistically significant, albeit small, advantage over Reid's Score in terms of discriminatory ability, as reflected in the AUC comparison. The results are reliable, as indicated by the confidence interval not including zero and the p-value being less than 0.05. The strong correlation between the two scores suggests that while they may differ slightly in performance, they are closely related in their measurement properties.

Reid’s Index (Cut Point at 4)

Recoded Data for Decision Test Statistics

	Pap Smear Positive	**Pap Smear Negative**	**Total**
Reid’s Positive	54	2	56
Reid’s Negative	12	232	244
Total	66	234	300

Table 11- Decision Test Statistics of Pap Smear Results with cut-off for Reid’s Index at 4

Among the 300 participants, 54 individuals with a Reid's Positive result also tested positive for a Pap smear, while only 2 individuals with a Reid's Positive result tested negative. This indicates that Reid's Positive results are associated with a high likelihood of a positive Pap smear outcome, as the vast majority (96.4%) of those identified as Reid's Positive also had positive Pap smears.

Conversely, among the 244 individuals categorized as Reid's Negative, 12 tested positive for a Pap smear, while the remaining 232 tested negative. This suggests that Reid's Negative results are generally indicative of a lower likelihood of cervical abnormalities, with only 4.9% of Reid's Negative individuals yielding a positive Pap smear result.

Overall, the distribution of results indicates that Reid's decision test is effective in identifying individuals at higher risk for cervical abnormalities. The majority of participants with Reid's Positive results correspond to positive Pap smear outcomes, while those with Reid's Negative results predominantly exhibit negative Pap smears.

χ^2 Tests			
	Value	**df**	**p**
χ^2	222	1	< .001
χ^2 continuity correction	217	1	< .001
Likelihood ratio	203	1	< .001
Fisher's exact test			< .001
N	300		

Table 12- Chi-Squared Tests for Pap Smear Results and Reid's Index Assessment

All tests indicate a highly significant association between Reid’s Score positivity and Pap smear positivity. The p-values < 0.001 suggest that the likelihood of observing these results under the null hypothesis is extremely low, leading to the rejection of the null hypothesis. This implies that there is a statistically significant relationship between the Reid’s Score positivity and Pap smear positivity in the studied population.

Comparative Measures			
		95% Confidence Intervals	
	Value	**Lower**	**Upper**
Odds ratio	522	113	2401

Table 13- Comparative Measures: Odds Ratio with 95% Confidence Intervals

The odds ratio is reported as 522, indicating that the odds of testing positive on a Pap smear are 522 times higher for individuals with a Reid's score classified as positive compared to those with a Reid's score classified as negative. This suggests a very strong association between a positive Reid's score and a positive Pap smear result, highlighting the effectiveness of Reid's score as a predictor of cervical abnormalities. The 95% confidence intervals range from 113 to 2401. This range indicates the precision of the odds ratio estimate. Since the confidence interval does not include 1, it confirms that the association is statistically significant. Although the wide range—from 113 to 2401—suggests variability in the estimate, the lower bound still indicates a strong likelihood of a significant association between Reid's score positivity and Pap smear positivity.

Overall, these findings suggest a substantial and statistically significant relationship between Reid's score positivity and the likelihood of a positive Pap smear result. This reinforces the utility of Reid's score in identifying individuals at increased risk for cervical abnormalities.

Test Accuracy	
	n
Total	300
Diseased	66
Healthy	234
Positive Tests	56
Negative Tests	244
True Test	286
Wrong Test	14

Table 14- Test Accuracy Metrics for Reid's Index Results

Total participants included 300 individuals. Out of these, 66 were diagnosed with the disease (indicated by positive Pap smear results), while 234 were identified as healthy (indicated by negative Pap smear results). The screening returned positive results for 56 participants and negative results for 244 participants. A total of 286 results accurately reflected the participants' health status, encompassing both true positives (correctly identified diseased individuals) and true negatives (correctly identified healthy individuals). There were 14 incorrect results, which include both false positives (healthy individuals incorrectly identified as diseased) and false negatives (diseased individuals incorrectly identified as healthy).

The data indicates a robust screening process with a high number of true test results, reflecting that the test is effective in distinguishing between healthy and diseased individuals. However, the presence of 14 wrong test results suggests that there is still room for improvement in the accuracy of the screening. The high proportion of correct results highlights the potential reliability of this test in clinical settings, although further investigation into the nature of the incorrect results would be necessary to refine the testing process and enhance its accuracy.

The accuracy of the test is approximately 95.33%, indicating that the screening process is highly effective in correctly identifying both diseased and healthy individuals. This high level of accuracy suggests that the test can be a reliable tool for cervical screening.

	Ratios
Sensitivity	81.8 %
Specificity	99.1 %
Accuracy	95.3 %
Prevalence	22.0 %
Positive Predictive Value	96.4 %

	Ratios
Negative Predictive Value	95.1 %
Post-test Disease Probability	96.4 %
Post-test Health Probability	95.1 %
Positive Likelihood Ratio	95.7
Negative Likelihood Ratio	0.183

Table 15- Test Accuracy Ratios and Performance Metrics for Reid's Index

Sensitivity is 81.8%, indicating that the test correctly identifies 81.8% of individuals who have the disease (true positives). This suggests a good ability to detect positive cases, although there is still a notable proportion of false negatives. Specificity is 99.1%, meaning the test effectively identifies healthy individuals, correctly classifying 99.1% of those without the disease (true negatives). This high value suggests that the test is excellent at ruling out healthy individuals.

Accuracy is 95.3%, reflecting the proportion of true results (both positive and negative) among all tests conducted. This indicates strong overall performance. Prevalence is 22.0%, showing that 22% of the tested population has the disease. This prevalence rate is important for understanding the context in which the test is applied.

Positive Predictive Value (PPV) is 96.4%, meaning that when the test is positive, there is a 96.4% chance that the individual actually has the disease. This highlights the reliability of positive test results. Negative Predictive Value (NPV) is 95.1%, indicating that when the test is negative, there is a 95.1% chance that the individual is truly healthy. This suggests high reliability for negative results as well.

Post-test Disease Probability is 96.4%, indicating the likelihood that an individual with a positive test result actually has the disease after considering the test results. Post-test Health Probability is 95.1%, reflecting the likelihood that an individual with a negative test result is truly healthy.

Positive Likelihood Ratio (LR+) is 95.7, indicating that a positive test result is very likely to occur in individuals with the disease compared to those without it, suggesting strong diagnostic capability for identifying disease. Negative Likelihood Ratio (LR-) is 0.183, suggesting that a negative test result is highly indicative of the absence of the disease, providing strong evidence against the disease when the test is negative.

Overall, the data indicates that the screening test has strong diagnostic performance, with high sensitivity, specificity, predictive values, and likelihood ratios, making it a valuable.

Swede’s Score (Cut Point at 5)

Recoded Data for Decision Test Statistics			
	Pap Smear Positive	**Pap Smear Negative**	**Total**
Swede’s Positive	66	7	73
Swede’s Negative	0	227	227
Total	66	234	300

Table 16- Decision Test Statistics of Pap Smear Results with cut-off for Swede’s Score at 5

Out of 300 individuals, all 66 with a positive Pap smear also had a positive Swede's score, indicating that a positive Swede’s Score reliably predicts a positive Pap smear. However, there are 7 individuals with a positive Swede's score who had a negative Pap smear, suggesting potential false positives in the Swede's score. Conversely, all 227 individuals with a negative Pap smear also had a negative Swede's score, reinforcing the correlation between the two tests. In total, 73 individuals had a positive Swede's score, while 227 had a negative score. All individuals with a positive Pap smear also have a positive Swede's score, indicating that a positive Pap smear is probably a reliable indicator of a positive Swede's score. However, there are 7 individuals with a positive Swede's score and a negative Pap smear, suggesting some instances of false positives in the Swede's score.

χ^2 Tests			
	Value	**df**	**p**
χ^2	263	1	< .001
χ^2 continuity correction	258	1	< .001
z test difference in 2 proportions	16.2		< .001
Likelihood ratio	270	1	< .001
Fisher's exact test			< .001
N	300		

Table 17- Chi-Squared Tests for Pap Smear Results and Swede's Score Assessment

All analyses indicate a strong and statistically significant association between Swede's score and Pap smear results ($p < 0.001$). Negative Swede's scores (≤4) are predominantly associated with negative Pap smears, while all positive Swede's scores (≥5) correspond to positive Pap smears. This consistent pattern suggests that Swede's score could be a valuable predictor of Pap smear outcomes. The findings reinforce the notion that elevated Swede's scores are linked to a higher likelihood of positive Pap smear results, further supporting the utility of Swede's score in cervical cancer screening and risk assessment.

Comparative Measures			
		95% Confidence Intervals	
	Value	**Lower**	**Upper**
Odds ratio	4034 *	227	71562

Comparative Measures			
		95% Confidence Intervals	
	Value	**Lower**	**Upper**
* Haldane-Anscombe correction applied			

Table 18- Comparative Measures: Odds Ratio w/ 95% Confidence Intervals (w/ Haldane-Anscombe Correction)

The odds ratio is reported as a high value (4034), indicating that the odds of testing positive on a Pap smear are significantly greater for individuals with a positive Swede's score (≥ 5) compared to those with a lower score. This suggests a very strong association between a positive Swede's score and a positive Pap smear result, underscoring the predictive power of the Swede's score in relation to cervical abnormalities. The large difference in proportions further illustrates this strong association, as the high odds ratios reflect the likelihood of a positive Pap smear in individuals with elevated Swede's scores. The application of the Haldane-Anscombe correction ensures that these estimates remain reliable, particularly in the context of small sample sizes or instances of zero counts.

Overall, these findings indicate a substantial and statistically significant relationship between a positive Swede's score and the likelihood of a positive Pap smear result. This reinforces the utility of the Swede's score as an effective tool for identifying individuals at increased risk for cervical abnormalities.

Test Accuracy	
	n
Total	300
Diseased	66
Healthy	234
Positive Tests	73
Negative Tests	227
True Test	293
Wrong Test	7

Table 19- Test Accuracy Metrics for Swede's Score

The data indicates that out of 300 individuals, the majority (234) are healthy, while 66 have the disease. The diagnostic test has a relatively high number of true test results (293), but there are still 7 incorrect classifications. The positive test results (73) indicate some overlap between true positives and false positives, and the high number of negative tests (227) reflects effective identification of healthy individuals. Overall, the test demonstrates a good level of accuracy (97.66%), but the presence of 7 wrong test results suggests that there is still room for improvement in diagnostic accuracy.

	Ratios
Sensitivity	100.0 %

	Ratios
Specificity	97.0 %
Accuracy	97.7 %
Prevalence	22.0 %
Positive Predictive Value	90.4 %
Negative Predictive Value	100.0 %
Post-test Disease Probability	90.4 %
Post-test Health Probability	100.0 %
Positive Likelihood Ratio	33.4
Negative Likelihood Ratio	0.00

Table 20- Test Accuracy Ratios and Performance Metrics for Swede's Score

Sensitivity is 100.0%, indicating that the test accurately identifies all individuals who have the disease (true positives), demonstrating an excellent ability to detect positive cases without any false negatives. Specificity is 97.0%, meaning the test effectively classifies healthy individuals, correctly identifying 97.0% of those without the disease (true negatives). This high specificity indicates that the test is very good at ruling out healthy individuals.

Accuracy is 97.66%, reflecting the proportion of true results (both positive and negative) among all tests conducted, indicating strong overall performance. Prevalence is 22.0%, suggesting that 22% of the tested population has the disease, which is important for understanding the context in which the test is applied.

Positive Predictive Value (PPV) is 90.4%, indicating that when the test is positive, there is a 90.4% chance that the individual actually has the disease. This highlights the reliability of positive test results. Negative Predictive Value (NPV) is 100.0%, suggesting that when the test is negative, the individual is guaranteed to be truly healthy, reflecting the test's high reliability for negative results.

Post-test Disease Probability is 90.4%, indicating the likelihood that an individual with a positive test result actually has the disease after considering the test results. Post-test Health Probability is 100.0%, reflecting the certainty that an individual with a negative test result is truly healthy.

Positive Likelihood Ratio (LR+) is 33.4, indicating that a positive test result is highly likely to occur in individuals with the disease compared to those without it, suggesting strong diagnostic capability for identifying disease. The Negative Likelihood Ratio (LR-) is 0.00, reinforcing that a negative test result effectively rules out the disease.

Overall, the data demonstrates that the screening test has exceptional diagnostic performance, with perfect sensitivity, high specificity, and robust predictive values, making it a highly valuable tool for assessing cervical abnormalities.

Cohen's kappa

			95% CI	
Ratings	kappa	SE	Lower	Upper
Average kappa	0.875			
Pap Smear - Reid's Score (Qualitative)	0.856	0.037	0.783	0.929
Pap Smear - Swede's Score (Qualitative)	0.935	0.024	0.887	0.982
Reid's Score (Qualitative) - Swede's Score (Qualitative)	0.833	0.039	0.757	0.909

Note. 300 subjects/items and 3 raters/measurements. Confidence intervals are asymptotic.

Table 21- Cohen's Kappa Values and Confidence Intervals for Inter-Rater Agreement in Pap Smear Assessments

The overall average kappa value was 0.875, indicating a high level of agreement among raters across all measurements. For the qualitative assessment of Pap smears using Reid's score, the kappa was 0.856 (SE = 0.037), with a 95% confidence interval (CI) ranging from 0.783 to 0.929. This suggests strong agreement between raters when employing this scoring system. In contrast, the qualitative assessment using Swede's score yielded a kappa of 0.935 (SE = 0.024), with a 95% CI of 0.887 to 0.982. This result reflects very strong agreement among raters, indicating high reliability for this scoring method. When comparing the Reid's score and Swede's score directly, the kappa value was 0.833 (SE = 0.039), with a 95% CI of 0.757 to 0.909. This indicates a strong level of agreement between the two qualitative scoring systems, although slightly lower than the individual scores.

Overall, the findings demonstrate a high degree of inter-rater reliability across the different scoring systems for Pap smear assessments, with the Swede's score exhibiting the strongest level of agreement.

Fleiss' kappa

			95% CI	
Ratings	Fleiss' kappa	SE	Lower	Upper
Overall	0.876	0.033	0.810	0.941
Negative	0.876	0.033	0.811	0.941
Positive	0.876	0.033	0.811	0.941

Note. 300 subjects/items and 3 raters/measurements. Confidence intervals are asymptotic. $p < 0.001$

Table 22- Fleiss' Kappa Values and Confidence Intervals for Inter-Rater Agreement in Pap Smear Assessments

The overall Fleiss' kappa value was 0.876 (SE = 0.033), indicating a high level of agreement among raters across all evaluations. This strong agreement surpasses what would be expected by chance, highlighting the consistency of the ratings. For both negative and positive ratings, the Fleiss' kappa was also 0.876 (SE = 0.033), demonstrating consistent inter-rater agreement across these categories. The 95% confidence intervals for all ratings ranged from 0.811 to 0.941, suggesting that we can be 95% confident that the true kappa values lie within this range. The p-value associated with the analysis was less than 0.001, indicating that the observed level of agreement is statistically significant.

Overall, these findings demonstrate a high degree of inter-rater reliability for both negative and positive assessments in Pap smear evaluations.

Discussion

The analysis included 300 participants, of whom 234 received negative Pap smear results and 66 received positive results, indicating a prevalence of 22% for cervical abnormalities. Age analysis revealed that the mean age for participants with negative results was slightly higher (44.9 years) compared to those with positive results (44.3 years), with both groups displaying relatively symmetrical age distributions. Notably, the age range for the negative group extended from 22 to 85 years, while the positive group's range was narrower, from 25 to 63 years.

In terms of parity, Multipara individuals represented the largest segment of the sample, with 70.7% achieving negative results. This contrasts with the Nullipara and Unipara groups, which showed a lower representation and a slightly higher prevalence of negative results. These findings suggest a potential correlation between parity and cervical health outcomes, warranting further investigation into how parity may influence the risk of abnormal Pap smear results.

The study also assessed contraceptive use, revealing that the majority of participants (70.7%) did not use contraception, with a notable percentage achieving negative Pap smear results. Conversely, only a small proportion of contraceptive users had positive results, indicating a potential protective effect of contraception against cervical abnormalities, though the small sample size limits definitive conclusions.

The performance of the both diagnostic tests (Reid’s Index & Swede’s Score) demonstrated high accuracy (95.33% & 97.66%, respectively), sensitivity (81.8% & 100%, respectively), and specificity (99.1% & 97.0%, respectively). Reid's Index and Swede's Score both showed strong associations with positive Pap smear results, with odds ratios of 522 and 4034, respectively. On ROC curve analysis, both tests performed remarkably with AUCs of 0.993 & 0.998, respectively. The optimal cut-points were 4 & 5, respectively, showcasing an optimal balance between Sensitivity & Specificity. Swede’s score showed a slight performance edge over Reid’s Index, which can be attributed to Swede’s Score having an additional criterion in its scoring system. These findings reinforce the utility of both scores as effective tools for predicting cervical abnormalities, particularly highlighting Swede's Score as having superior diagnostic performance. Overall, the results suggest that these screening methods are reliable

in identifying individuals at increased risk for cervical disease, emphasizing the importance of continued research in this area.

The initial analysis of Pap smear results revealed critical insights into the demographics and health outcomes of the participants, with a total of 300 individuals evaluated. A significant finding was that 78% of the participants received negative Pap smear results, indicating no significant cervical abnormalities. The study noted that among those with positive results, the majority were Multipara individuals, suggesting a potential correlation between parity and cervical health. Additionally, the analysis highlighted that, the individuals who do not use contraception were more prevalent in the negative results category, although a notable proportion still tested positive. This suggests that contraceptive use may have a protective factor, warranting further investigation.

Moreover, the findings indicated strong associations between Reid's and Swede's Scores and the likelihood of positive Pap smear results. For instance, individuals classified as Reid's Positive showed a 96.4% likelihood of having a positive Pap smear, underscoring the predictive power of these indices in identifying cervical abnormalities. The analysis further established that Swede's Score demonstrated even higher performance metrics, with an odds ratio of 4034 indicating a very strong association with positive outcomes.

In contrast, the Modified Swede Colposcopic Index (MSCI) (Swede's Score) and Modified Reid Index (MRI) (Reid's Index) focus specifically on enhancing colposcopic evaluations, refining the diagnostic process for cervical lesions. These indices are designed to standardize the interpretation of colposcopic findings, which can often be subjective. The Swede's Score improves upon the Reid's Index by incorporating critical elements such as lesion size and location, thereby providing a clearer framework for assessing disease severity. Research indicates that larger lesions are more likely to harbor high-grade lesions, making this a vital factor for accurate diagnosis.

The Swede Score's structured criteria allow clinicians to differentiate between lesion types effectively. For example, dense, opaque acetowhite staining suggests high-grade lesions (CIN2+), while lighter staining indicates lower-grade changes. In contrast, the Reid's Index retains a focus on color, margins, and vascular patterns, utilizing observable characteristics to inform diagnoses. The emphasis on margin characteristics in both indices enables a more nuanced understanding of lesion severity, with irregular or jagged margins associated with a higher likelihood of significant disease.

Importantly, performance metrics reveal that the Swede's Score achieves an impressive 100% sensitivity for detecting high-grade lesions at a threshold score of 5, compared to the Reid's Index sensitivity of 83.7% at the same threshold. While the MRI can achieve higher specificity at increased thresholds, this comes at the expense of sensitivity, making it less suitable for initial screening. The Swede Score's flexibility in cut-offs allows for adaptability in various clinical contexts, maximizing both sensitivity and specificity based on the clinical objectives.

The findings of this study align closely with previous research in the field of cervical cancer screening and diagnosis, particularly in terms of the predictive power of various indices. Similar to earlier studies, such as those by Bowring et al. (2010) and Rahman et al. (2020), our results reinforce the significance of structured assessment tools like the Reid's Index and Swede's Scores in identifying cervical abnormalities. Notably, the high likelihood of positive

Pap smear results among individuals classified as Reid's Positive mirrors findings from the literature, highlighting the index's effectiveness in predicting high-grade lesions. Furthermore, our study's emphasis on the association between multiparity and positive results echoes previous conclusions regarding the demographic factors influencing cervical health. The superior performance metrics of the Swede's Score compared to the Reid's Index also corroborate findings from Ranga et al. (2017) and Priya et al. (2024), which demonstrated the Swede's Scores enhanced sensitivity and practicality in clinical settings. Collectively, these comparisons underline the consistency of our findings with established research, further validating the importance of using standardized indices in cervical cancer screening to improve diagnostic accuracy and patient outcomes.

When evaluating Pap smears using Reid's score, a kappa of 0.856 (SE = 0.037) was observed, with a 95% confidence interval (CI) ranging from 0.783 to 0.929. This finding underscores the strong agreement between raters utilizing this scoring system. In contrast, the Swede's score demonstrated an even higher kappa value of 0.935 (SE = 0.024), with a 95% CI of 0.887 to 0.982, reflecting very strong inter-rater agreement and indicating high reliability for this method. The comparison between Reid's and Swede's scores yielded a kappa of 0.833 (SE = 0.039) and a 95% CI of 0.757 to 0.909, indicating a strong level of agreement between the two qualitative scoring systems, although slightly lower than the individual scores.

In addition, the overall Fleiss' kappa value of 0.876 (SE = 0.033) further corroborates the high level of agreement among raters across all evaluations. This consistent agreement exceeds what would be expected by chance, highlighting the reliability of the ratings across different categories. Notably, both negative and positive ratings yielded a Fleiss' kappa of 0.876 (SE = 0.033), with 95% confidence intervals ranging from 0.811 to 0.941. This suggests that we can be 95% confident that the true kappa values lie within this range. The statistically significant p-value of less than 0.001 reinforces the robustness of these findings.

Overall, these results indicate a high degree of inter-rater reliability for both negative and positive assessments in Pap smear evaluations. The Swede's score stands out as the most reliable scoring method among those assessed, demonstrating the potential for enhanced consistency in clinical practice.

The initial analysis and the advancements represented by the Swede's Score and Reid's Index underscore the evolving landscape of cervical cancer screening. The initial findings demonstrate the effectiveness of traditional Pap smear screening methods, particularly when augmented by predictive indices like Reid's Index and Swede's Scores. Meanwhile, the Swede's Score and Reid's Index enhance diagnostic accuracy in colposcopy through structured, standardized assessments that account for critical lesion characteristics. By integrating these approaches, clinicians can develop a more comprehensive and effective cervical screening strategy, ultimately improving early detection and treatment of cervical abnormalities. This dual focus not only supports the ongoing effort to reduce cervical cancer incidence but also ensures that patients receive timely and accurate diagnoses, thereby improving overall health outcomes.

The future prospects of this study suggest a multifaceted approach to enhancing cervical cancer screening and management, encompassing technological, educational, and collaborative strategies. One of the most exciting prospects lies in leveraging advanced technologies such as artificial intelligence (AI) and machine learning. These technologies can analyze vast amounts

of data from colposcopic images and patient histories to identify subtle patterns and predictive factors that human evaluators might overlook. For instance, AI algorithms could assist in automating the scoring of indices like the Swede's Score and Reid's Index, thereby reducing variability and increasing consistency in assessments. This could lead to more accurate diagnoses and enable earlier intervention for high-grade lesions.

To ensure that the Swede's Score and Reid's Index are effective in a wide range of clinical settings, future research should focus on validating these tools across diverse populations. This involves conducting multicenter studies that incorporate various demographic factors, such as age, ethnicity, and socioeconomic status. By understanding how these indices perform in different contexts, we can refine their criteria to enhance sensitivity and specificity for various groups, ultimately leading to more equitable healthcare outcomes.

In summary, the future of cervical cancer screening, guided by the findings of this study, holds significant promise. Through technological advancements, expanded applicability, continuous refinement of diagnostic tools, enhanced education for healthcare providers, collaborative efforts, and a strong focus on early detection, we can continue to improve patient outcomes and reduce the global burden of cervical cancer.

Limitations of the Study

This study, while providing valuable insights into the effectiveness of Reid's Score and Swede's Score in predicting cervical abnormalities, has several limitations that warrant consideration. First, the sample size, although adequate at 300 participants, may not fully represent the broader population, potentially limiting the generalizability of the findings. The predominance of Multipara individuals in the sample could skew results, as their demographic characteristics may not reflect those of Nullipara and Unipara groups, which are underrepresented.

Second, the reliance on Pap smear results as the primary diagnostic criterion may introduce biases. Pap smears, while widely used, can have varying sensitivity and specificity, is mostly reporter dependent on classifying the lesions, leading to potential misclassifications. Additionally, the study did not account for potential confounding factors such as HPV status, or smoking, which could influence screening outcomes and risk assessments.

Another limitation is the cross-sectional nature of the study, which does not allow for longitudinal tracking of participant health outcomes over time. This limits the ability to draw causal inferences about the relationship between scoring indices and the development of cervical lesions.

Finally, the study's design may not have included a diverse enough range of clinical settings, which could affect the applicability of the findings in different healthcare environments. This limitation underscores the need for further research across varied populations and settings to validate and enhance the applicability of the results. Addressing these limitations in future studies will be crucial for strengthening the evidence base surrounding cervical cancer screening and diagnosis.

Future Prospects of the Study

The field of cervical cancer screening is continuously evolving, and there is a need for ongoing refinement of scoring systems based on emerging research. Future studies should focus on identifying additional criteria that may improve the predictive power of existing indices. Incorporating new findings, such as insights from genomic studies or the impact of lifestyle factors on cervical health, can help in developing a more comprehensive diagnostic tool. Regular updates to these indices would ensure they remain relevant and effective as new evidence emerges.

Enhancing the training and education of healthcare providers is crucial for the effective implementation of standardized indices in clinical practice. Future initiatives could include developing comprehensive training programs that focus on the use of these tools, interpretation of results, and decision-making processes in cervical cancer management. By equipping clinicians with the knowledge and skills to utilize these indices effectively, we can improve the quality of care and diagnostic accuracy.

Collaboration between researchers, clinicians, and public health organizations is essential for translating research findings into practice. Building partnerships can facilitate the sharing of best practices, resources, and data, which can enhance the overall effectiveness of cervical cancer screening programs. Moreover, engaging with policymakers to integrate these advancements into national screening guidelines will help standardize practices and ensure that high-quality screening is accessible to all populations.

Ultimately, the overarching goal of these advancements is to promote early detection and timely intervention for cervical abnormalities. By improving the accuracy and accessibility of screening tools, we can significantly reduce the incidence of cervical cancer. Future research should also explore the implementation of these screening tools in low-resource settings, ensuring that even underserved populations have access to effective cervical cancer prevention strategies.

Conclusion

In conclusion, this study reinforces the vital role of structured assessment tools, such as the Swede’s Score and Reid's Index, in enhancing the efficacy of cervical cancer screening and diagnosis. The findings demonstrate that these standardized indices significantly improve diagnostic accuracy, allowing clinicians to make more informed decisions regarding patient care. Our results align closely with existing literature, which emphasizes the importance of these indices in reliably identifying high-grade lesions and enhancing overall patient outcomes. This consistency with previous studies underscores the need for continued adoption and integration of these methodologies in clinical practice.

Looking toward the future, the prospects for cervical cancer screening and management are promising. Advancements in technology, particularly in artificial intelligence and machine learning, hold the potential to revolutionize how we interpret colposcopic findings. These innovations can provide clinicians with advanced diagnostic tools that augment traditional methods, increasing accuracy and efficiency. Additionally, ongoing research into the biological and genetic markers of cervical lesions may further refine screening processes, allowing for more personalized approaches to patient care.

Moreover, fostering collaboration among healthcare professionals, researchers, and policymakers is essential in promoting equitable access to effective screening tools. Ensuring that these advanced methodologies are available in low-resource settings will be critical in addressing disparities in cervical cancer incidence and outcomes. Public health initiatives that raise awareness and encourage regular screening can significantly contribute to early detection and timely intervention, ultimately leading to reduced mortality rates.

In brief, our collective efforts to enhance cervical cancer screening not only focus on refining diagnostic practices but also aim to create a more inclusive healthcare environment. By embracing advancements and fostering collaboration, we can ensure that effective screening tools reach diverse populations. This comprehensive approach will ultimately promote early detection and timely management, significantly reducing the incidence of cervical cancer and improving the overall quality of care for patients. Through these initiatives, we move closer to a future where cervical cancer is effectively prevented and managed, ensuring better health outcomes for all.

References

- Adsul, P. *et al.* (2017) 'Implementing community-based cervical cancer screening programs using visual inspection with acetic acid in India: A systematic review', *Cancer Epidemiology*, 49, pp. 161–174. Available at: https://doi.org/10.1016/j.canep.2017.06.008.
- Alan, M. *et al.* (2020) 'Correlation of Swede score colposcopy scoring system and histopathological results in patients with high-risk HPV infection other than HPV16 and 18', *International Journal of Gynecological Cancer: Official Journal of the International Gynecological Cancer Society*, 30(1), pp. 35–40. Available at: https://doi.org/10.1136/ijgc-2019-000932.
- Ali, M. *et al.* (2024) 'HPV DNA status and clinical history of patients are supplements for accurate reporting of the cytological Pap smear', *Scientific Reports*, 14(1), p. 17486. Available at: https://doi.org/10.1038/s41598-024-68344-4.
- Alzahrani, A.S. (2019) 'PI3K/Akt/mTOR inhibitors in cancer: At the bench and bedside', *Seminars in Cancer Biology*, 59, pp. 125–132. Available at: https://doi.org/10.1016/j.semcancer.2019.07.009.
- Araldi, R.P. *et al.* (2018) 'The human papillomavirus (HPV)-related cancer biology: An overview', *Biomedicine & Pharmacotherapy = Biomedecine & Pharmacotherapie*, 106, pp. 1537–1556. Available at: https://doi.org/10.1016/j.biopha.2018.06.149.
- Arbyn, M. *et al.* (2022) 'Accuracy and effectiveness of HPV mRNA testing in cervical cancer screening: a systematic review and meta-analysis', *The Lancet. Oncology*, 23(7), pp. 950–960. Available at: https://doi.org/10.1016/S1470-2045(22)00294-7.
- Aue-Aungkul, A. and Suprasert, P. (2015) 'Reid Colposcopic Index Evaluation: Comparison of General and Oncologic Gynecologists', *Asian Pacific journal of cancer prevention: APJCP*, 16(12), pp. 5001–5004. Available at: https://doi.org/10.7314/apjcp.2015.16.12.5001.
- Bello, J.O.M. *et al.* (2015) 'Regulation of the Wnt/β-Catenin Signaling Pathway by Human Papillomavirus E6 and E7 Oncoproteins', *Viruses*, 7(8), pp. 4734–4755. Available at: https://doi.org/10.3390/v7082842.
- Bodily, J.M., Mehta, K.P.M. and Laimins, L.A. (2011) 'Human papillomavirus E7 enhances hypoxia-inducible factor 1-mediated transcription by inhibiting binding of histone deacetylases', *Cancer Research*, 71(3), pp. 1187–1195. Available at: https://doi.org/10.1158/0008-5472.CAN-10-2626.
- Boonlikit, S. (2016) 'Performance of the abbreviated Reid colposcopic index in prediction of high-grade lesions', *International Journal of Gynecology & Obstetrics*, 134(1), pp. 41–44. Available at: https://doi.org/10.1016/j.ijgo.2015.10.025.
- Bossler, F., Hoppe-Seyler, K. and Hoppe-Seyler, F. (2019) 'PI3K/AKT/mTOR Signaling Regulates the Virus/Host Cell Crosstalk in HPV-Positive Cervical Cancer Cells', *International Journal of Molecular Sciences*, 20(9), p. 2188. Available at: https://doi.org/10.3390/ijms20092188.
- Bowring, J. *et al.* (2010) 'The Swede score: evaluation of a scoring system designed to improve the predictive value of colposcopy', *Journal of Lower Genital Tract Disease*, 14(4), pp. 301–305. Available at: https://doi.org/10.1097/LGT.0b013e3181d77756.
- Burness, J.V., Schroeder, J.M. and Warren, J.B. (2020) 'Cervical Colposcopy: Indications and Risk Assessment', *American Family Physician*, 102(1), pp. 39–48.
- Buskwofie, A., David-West, G. and Clare, C.A. (2020) 'A Review of Cervical Cancer: Incidence and Disparities', *Journal of the National Medical Association*, 112(2), pp. 229–232. Available at: https://doi.org/10.1016/j.jnma.2020.03.002.

- Caldeira, S., Dong, W. and Tommasino, M. (2005) 'Analysis of E7/Rb associations', *Methods in Molecular Medicine*, 119, pp. 363–379. Available at: https://doi.org/10.1385/1-59259-982-6:363.
- Chatterjee, S. *et al.* (2016) 'HPV and Cervical Cancer Epidemiology - Current Status of HPV Vaccination in India', *Asian Pacific journal of cancer prevention: APJCP*, 17(8), pp. 3663–3673.
- Costa, S. *et al.* (2000) 'Combined Pap smear, cervicography and HPV DNA testing in the detection of cervical intraepithelial neoplasia and cancer', *Acta Cytologica*, 44(3), pp. 310–318. Available at: https://doi.org/10.1159/000328471.
- Donmez, H.G. *et al.* (2022) 'Impact of Human Papillomavirus on Wnt/Beta-Catenin Signaling in Morphological Inconspicuous Cervicovaginal Cells', *Acta Cytologica*, 66(5), pp. 409–419. Available at: https://doi.org/10.1159/000522635.
- Drews, C.M., Case, S. and Vande Pol, S.B. (2019) 'E6 proteins from high-risk HPV, low-risk HPV, and animal papillomaviruses activate the Wnt/β-catenin pathway through E6AP-dependent degradation of NHERF1', *PLoS pathogens*, 15(4), p. e1007575. Available at: https://doi.org/10.1371/journal.ppat.1007575.
- Driscoll, S.D. *et al.* (2018) 'Accuracy of visual inspection performed by community health workers in cervical cancer screening', *International Journal of Gynaecology and Obstetrics: The Official Organ of the International Federation of Gynaecology and Obstetrics*, 142(3), pp. 260–269. Available at: https://doi.org/10.1002/ijgo.12535.
- Estêvão, D. *et al.* (2019) 'Hallmarks of HPV carcinogenesis: The role of E6, E7 and E5 oncoproteins in cellular malignancy', *Biochimica Et Biophysica Acta. Gene Regulatory Mechanisms*, 1862(2), pp. 153–162. Available at: https://doi.org/10.1016/j.bbagrm.2019.01.001.
- Fernandes, A. *et al.* (2022) 'Human papillomavirus-independent cervical cancer', *International Journal of Gynecological Cancer: Official Journal of the International Gynecological Cancer Society*, 32(1), pp. 1–7. Available at: https://doi.org/10.1136/ijgc-2021-003014.
- Fernandes, J.V. *et al.* (2015) 'Link between chronic inflammation and human papillomavirus-induced carcinogenesis (Review)', *Oncology Letters*, 9(3), pp. 1015–1026. Available at: https://doi.org/10.3892/ol.2015.2884.
- Funk, J.O. *et al.* (1997) 'Inhibition of CDK activity and PCNA-dependent DNA replication by p21 is blocked by interaction with the HPV-16 E7 oncoprotein', *Genes & Development*, 11(16), pp. 2090–2100. Available at: https://doi.org/10.1101/gad.11.16.2090.
- Garzón-Olivares, C.D. *et al.* (2022) 'Primary HPV-DNA screening in women under 30 years of age: health technology assessment', *Revista Colombiana De Obstetricia Y Ginecologia*, 73(2), pp. 203–222. Available at: https://doi.org/10.18597/rcog.3866.
- Gavinski, K. and DiNardo, D. (2023) 'Cervical Cancer Screening', *The Medical Clinics of North America*, 107(2), pp. 259–269. Available at: https://doi.org/10.1016/j.mcna.2022.10.006.
- Georgescu, S.R. *et al.* (2018) 'New Insights in the Pathogenesis of HPV Infection and the Associated Carcinogenic Processes: The Role of Chronic Inflammation and Oxidative Stress', *Journal of Immunology Research*, 2018, p. 5315816. Available at: https://doi.org/10.1155/2018/5315816.
- Ghafouri-Fard, S. *et al.* (2022) 'The interaction between human papilloma viruses related cancers and non-coding RNAs', *Pathology, Research and Practice*, 234, p. 153939. Available at: https://doi.org/10.1016/j.prp.2022.153939.

- Glaviano, A. *et al.* (2023) 'PI3K/AKT/mTOR signaling transduction pathway and targeted therapies in cancer', *Molecular Cancer*, 22(1), p. 138. Available at: https://doi.org/10.1186/s12943-023-01827-6.
- Graham, S.V. (2017) 'The human papillomavirus replication cycle, and its links to cancer progression: a comprehensive review', *Clinical Science (London, England: 1979)*, 131(17), pp. 2201–2221. Available at: https://doi.org/10.1042/CS20160786.
- He, G. *et al.* (2021) 'microRNA-21: a key modulator in oncogenic viral infections', *RNA biology*, 18(5), pp. 809–817. Available at: https://doi.org/10.1080/15476286.2021.1880756.
- Henken, F.E. *et al.* (2012) 'The functional role of Notch signaling in HPV-mediated transformation is dose-dependent and linked to AP-1 alterations', *Cellular Oncology (Dordrecht, Netherlands)*, 35(2), pp. 77–84. Available at: https://doi.org/10.1007/s13402-011-0062-4.
- Hong, D.G. *et al.* (2010) 'Prediction of high-grade squamous intraepithelial lesions using the modified Reid index', *International Journal of Clinical Oncology*, 15(1), pp. 65–69. Available at: https://doi.org/10.1007/s10147-009-0012-6.
- Hoppe-Seyler, K. *et al.* (2018) 'The HPV E6/E7 Oncogenes: Key Factors for Viral Carcinogenesis and Therapeutic Targets', *Trends in Microbiology*, 26(2), pp. 158–168. Available at: https://doi.org/10.1016/j.tim.2017.07.007.
- Huang, J. *et al.* (2022) 'Global distribution, risk factors, and recent trends for cervical cancer: A worldwide country-level analysis', *Gynecologic Oncology*, 164(1), pp. 85–92. Available at: https://doi.org/10.1016/j.ygyno.2021.11.005.
- International Agency for Research on Cancer (IARC) (n.d.) (IARC n.d.) Colposcopy Atlas. Available at: https://screening.iarc.fr/atlascolpo.php (Accessed: 14 October 2024).
- Ito, K. *et al.* (2020) 'A comparison of liquid-based and conventional cytology using data for cervical cancer screening from the Japan Cancer Society', *Japanese Journal of Clinical Oncology*, 50(2), pp. 138–144. Available at: https://doi.org/10.1093/jjco/hyz161.
- Johansson, C. *et al.* (2012) 'HPV-16 E2 contributes to induction of HPV-16 late gene expression by inhibiting early polyadenylation', *The EMBO journal*, 31(14), pp. 3212–3227. Available at: https://doi.org/10.1038/emboj.2012.147.
- Johnson, C.A. *et al.* (2019) 'Cervical Cancer: An Overview of Pathophysiology and Management', *Seminars in Oncology Nursing*, 35(2), pp. 166–174. Available at: https://doi.org/10.1016/j.soncn.2019.02.003.
- Jose, L. *et al.* (2023) 'Focal Adhesion Kinase Binds to the HPV E2 Protein to Regulate Initial Replication after Infection', *Pathogens (Basel, Switzerland)*, 12(10), p. 1203. Available at: https://doi.org/10.3390/pathogens12101203.
- Karar, J. and Maity, A. (2011) 'PI3K/AKT/mTOR Pathway in Angiogenesis', *Frontiers in Molecular Neuroscience*, 4, p. 51. Available at: https://doi.org/10.3389/fnmol.2011.00051.
- Kim, Y.T. and Zhao, M. (2005) 'Aberrant cell cycle regulation in cervical carcinoma', *Yonsei Medical Journal*, 46(5), pp. 597–613. Available at: https://doi.org/10.3349/ymj.2005.46.5.597.
- von Knebel Doeberitz, M. *et al.* (2012) 'Biomarkers for cervical cancer screening: the role of p16(INK4a) to highlight transforming HPV infections', *Expert Review of Proteomics*, 9(2), pp. 149–163. Available at: https://doi.org/10.1586/epr.12.13.
- Krishnan, A. *et al.* (2021) 'Preparedness of primary and secondary health facilities in India to address major noncommunicable diseases: results of a National Noncommunicable Disease Monitoring Survey (NNMS)', *BMC health services research*, 21(1), p. 757. Available at: https://doi.org/10.1186/s12913-021-06530-0.

- Kudela, E. *et al.* (2020) 'Colposcopic scoring indexes in the evaluation of cervical lesions with the cytological result of atypical squamous cells, cannot exclude high-grade lesion', *Journal of Obstetrics and Gynaecology Research*, 46(2), pp. 314–319. Available at: https://doi.org/10.1111/jog.14158.
- Lee, H., Kang, Y. and Ju, W. (2016) 'Cervical Cancer Screening in Developing Countries: Using Visual Inspection Methods', *Clinical Journal of Oncology Nursing*, 20(1), pp. 79–83. Available at: https://doi.org/10.1188/16.CJON.79-83.
- Lee, S.Y. *et al.* (2013) 'Histone deacetylase inhibitor AR-42 enhances E7-specific $CD8^+$ T cell-mediated antitumor immunity induced by therapeutic HPV DNA vaccination', *Journal of Molecular Medicine (Berlin, Germany)*, 91(10), pp. 1221–1231. Available at: https://doi.org/10.1007/s00109-013-1054-9.
- Li, S. *et al.* (2019) 'Ubiquitination of the HPV Oncoprotein E6 Is Critical for E6/E6AP-Mediated p53 Degradation', *Frontiers in Microbiology*, 10, p. 2483. Available at: https://doi.org/10.3389/fmicb.2019.02483.
- Li, X.-Y. *et al.* (2023) 'Non-Genetic Factors and Risk of Cervical Cancer: An Umbrella Review of Systematic Reviews and Meta-Analyses of Observational Studies', *International Journal of Public Health*, 68, p. 1605198. Available at: https://doi.org/10.3389/ijph.2023.1605198.
- Lim, E.H. *et al.* (2010) 'Cervical dysplasia: assessing methylation status (Methylight) of CCNA1, DAPK1, HS3ST2, PAX1 and TFPI2 to improve diagnostic accuracy', *Gynecologic Oncology*, 119(2), pp. 225–231. Available at: https://doi.org/10.1016/j.ygyno.2010.07.028.
- Ling, J. *et al.* (2022) 'Human papillomavirus 16 E6/E7 contributes to immune escape and progression of cervical cancer by regulating miR-142-5p/PD-L1 axis', *Archives of Biochemistry and Biophysics*, 731, p. 109449. Available at: https://doi.org/10.1016/j.abb.2022.109449.
- Liu, J. *et al.* (2022) 'Wnt/β-catenin signalling: function, biological mechanisms, and therapeutic opportunities', *Signal Transduction and Targeted Therapy*, 7(1), p. 3. Available at: https://doi.org/10.1038/s41392-021-00762-6.
- Manning, B.D. and Toker, A. (2017) 'AKT/PKB Signaling: Navigating the Network', *Cell*, 169(3), pp. 381–405. Available at: https://doi.org/10.1016/j.cell.2017.04.001.
- Manzo-Merino, J. *et al.* (2013) 'HPV E6 oncoprotein as a potential therapeutic target in HPV related cancers', *Expert Opinion on Therapeutic Targets*, 17(11), pp. 1357–1368. Available at: https://doi.org/10.1517/14728222.2013.832204.
- Mitra, A. *et al.* (2016) 'Cervical intraepithelial neoplasia: screening and management', *British Journal of Hospital Medicine (London, England: 2005)*, 77(8), pp. C118-123. Available at: https://doi.org/10.12968/hmed.2016.77.8.C118.
- Munger, K., Gwin, T.K. and McLaughlin-Drubin, M.E. (2013) 'p16 in HPV-associated cancers', *Oncotarget*, 4(11), pp. 1864–1865. Available at: https://doi.org/10.18632/oncotarget.1523.
- Nelson, C.W. and Mirabello, L. (2023) 'Human papillomavirus genomics: Understanding carcinogenicity', *Tumour Virus Research*, 15, p. 200258. Available at: https://doi.org/10.1016/j.tvr.2023.200258.
- Nero, C. *et al.* (2019) 'PTEN and Gynecological Cancers', *Cancers*, 11(10), p. 1458. Available at: https://doi.org/10.3390/cancers11101458.
- Nitulescu, G.M. *et al.* (2018) 'The Akt pathway in oncology therapy and beyond (Review)', *International Journal of Oncology*, 53(6), pp. 2319–2331. Available at: https://doi.org/10.3892/ijo.2018.4597.

- Nkwabong, E., Laure Bessi Badjan, I. and Sando, Z. (2019) 'Pap smear accuracy for the diagnosis of cervical precancerous lesions', *Tropical Doctor*, 49(1), pp. 34–39. Available at: https://doi.org/10.1177/0049475518798532.
- Olusola, P. *et al.* (2019) 'Human Papilloma Virus-Associated Cervical Cancer and Health Disparities', *Cells*, 8(6), p. 622. Available at: https://doi.org/10.3390/cells8060622.
- Oyouni, A.A.A. (2023) 'Human papillomavirus in cancer: Infection, disease transmission, and progress in vaccines', *Journal of Infection and Public Health*, 16(4), pp. 626–631. Available at: https://doi.org/10.1016/j.jiph.2023.02.014.
- Pal, A. and Kundu, R. (2019) 'Human Papillomavirus E6 and E7: The Cervical Cancer Hallmarks and Targets for Therapy', *Frontiers in Microbiology*, 10, p. 3116. Available at: https://doi.org/10.3389/fmicb.2019.03116.
- Phaliwong, P. *et al.* (2018) 'Cervical Cancer Detection between Conventional and Liquid Based Cervical Cytology: a 6-Year Experience in Northern Bangkok Thailand', *Asian Pacific journal of cancer prevention: APJCP*, 19(5), pp. 1331–1336. Available at: https://doi.org/10.22034/APJCP.2018.19.5.1331.
- Priya, Dr.S. *et al.* (2024) 'Modified swede colposcopic index versus modified reid index in colposcopic screening for premalignant & malignant lesions of the cervix: a cross-sectional analysis', *Cancer Treatment and Research Communications*, 40, p. 100828. Available at: https://doi.org/10.1016/j.ctarc.2024.100828.
- Rahman, Z., Yadav, G. and Tripathi, U. (2020) 'The Diagnostic Efficacy of Swede Score for Prediction of Pre-invasive Cervical Lesions: A Prospective Hospital-Based Study', *The Journal of Obstetrics and Gynecology of India*, 70(6), pp. 497–502. Available at: https://doi.org/10.1007/s13224-020-01344-2.
- Rajaram, S. and Gupta, B. (2021) 'Screening for cervical cancer: Choices & dilemmas', *The Indian Journal of Medical Research*, 154(2), pp. 210–220. Available at: https://doi.org/10.4103/ijmr.IJMR_857_20.
- Ranga, R. *et al.* (2017) 'A Comparison of the Strength of Association of Reid Colposcopic Index and Swede Score With Cervical Histology', *Journal of Lower Genital Tract Disease*, 21(1), pp. 55–58. Available at: https://doi.org/10.1097/LGT.0000000000000278.
- Rodrigues, C. *et al.* (2019) 'Notch signalling in cervical cancer', *Experimental Cell Research*, 385(2), p. 111682. Available at: https://doi.org/10.1016/j.yexcr.2019.111682.
- Rossi, P.G. and Rebolj, M. (2022) 'HPV mRNA testing in cervical cancer screening', *The Lancet. Oncology*, 23(10), p. e436. Available at: https://doi.org/10.1016/S1470-2045(22)00486-7.
- Shen-Gunther, J. *et al.* (2016) 'Molecular Pap smear: HPV genotype and DNA methylation of ADCY8, CDH8, and ZNF582 as an integrated biomarker for high-grade cervical cytology', *Clinical Epigenetics*, 8(1), p. 96. Available at: https://doi.org/10.1186/s13148-016-0263-9.
- Shrestha, A.D. *et al.* (2018) 'Cervical Cancer Prevalence, Incidence and Mortality in Low and Middle Income Countries: A Systematic Review', *Asian Pacific journal of cancer prevention: APJCP*, 19(2), pp. 319–324. Available at: https://doi.org/10.22034/APJCP.2018.19.2.319.
- Shukla, P. *et al.* (2023) 'Readiness and Motivation of ASHAs towards Their Participation in Non-Communicable Disease Control Programmein North India:A Cross Sectional Study', *Asian Pacific journal of cancer prevention: APJCP*, 24(9), pp. 3235–3241. Available at: https://doi.org/10.31557/APJCP.2023.24.9.3235.
- Sivars, L. *et al.* (2023) 'The current status of cell-free human papillomavirus DNA as a biomarker in cervical cancer and other HPV-associated tumors: A review', *International*

Journal of Cancer, 152(11), pp. 2232–2242. Available at: https://doi.org/10.1002/ijc.34333.

- Small, W. *et al.* (2017) 'Cervical cancer: A global health crisis', *Cancer*, 123(13), pp. 2404–2412. Available at: https://doi.org/10.1002/cncr.30667.
- Sompawong, N. *et al.* (2019) 'Automated Pap Smear Cervical Cancer Screening Using Deep Learning', *Annual International Conference of the IEEE Engineering in Medicine and Biology Society. IEEE Engineering in Medicine and Biology Society. Annual International Conference*, 2019, pp. 7044–7048. Available at: https://doi.org/10.1109/EMBC.2019.8856369.
- Song, B. *et al.* (2015) 'Inhibition of Notch Signaling Promotes the Adipogenic Differentiation of Mesenchymal Stem Cells Through Autophagy Activation and PTEN-PI3K/AKT/mTOR Pathway', *Cellular Physiology and Biochemistry: International Journal of Experimental Cellular Physiology, Biochemistry, and Pharmacology*, 36(5), pp. 1991–2002. Available at: https://doi.org/10.1159/000430167.
- Sreedevi, A., Javed, R. and Dinesh, A. (2015) 'Epidemiology of cervical cancer with special focus on India', *International Journal of Women's Health*, 7, pp. 405–414. Available at: https://doi.org/10.2147/IJWH.S50001.
- Suwanthananon, C. and Inthasorn, P. (2020) 'A comparison of the associations of Reid Colposcopic Index and Swede Score with cervical histology', *The Journal of Obstetrics and Gynaecology Research*, 46(4), pp. 618–624. Available at: https://doi.org/10.1111/jog.14200.
- Szymonowicz, K.A. and Chen, J. (2020) 'Biological and clinical aspects of HPV-related cancers', *Cancer Biology & Medicine*, 17(4), pp. 864–878. Available at: https://doi.org/10.20892/j.issn.2095-3941.2020.0370.
- Taneja, N. *et al.* (2021) 'Knowledge, Attitude, and Practice on Cervical Cancer and Screening Among Women in India: A Review', *Cancer Control: Journal of the Moffitt Cancer Center*, 28, p. 10732748211010799. Available at: https://doi.org/10.1177/10732748211010799.
- del Toro-Arreola, S., García-Chagollán, M. and Jave-Suárez, L.F. (2015) '[Escape mechanisms to the innate immune response in HPV-associated cervical cancer]', *Revista Medica Del Instituto Mexicano Del Seguro Social*, 53 Suppl 2, pp. S194-199.
- Travé, G. and Zanier, K. (2016) 'HPV-mediated inactivation of tumor suppressor p53', *Cell Cycle (Georgetown, Tex.)*, 15(17), pp. 2231–2232. Available at: https://doi.org/10.1080/15384101.2016.1191257.
- Tsikouras, P. *et al.* (2016) 'Cervical cancer: screening, diagnosis and staging', *Journal of B.U.ON.: official journal of the Balkan Union of Oncology*, 21(2), pp. 320–325.
- Tzenov, Y.R. *et al.* (2013) 'Human papilloma virus (HPV) E7-mediated attenuation of retinoblastoma (Rb) induces hPygopus2 expression via Elf-1 in cervical cancer', *Molecular cancer research: MCR*, 11(1), pp. 19–30. Available at: https://doi.org/10.1158/1541-7786.MCR-12-0510.
- Vikraman, S.M., Khanna, D. and Dandpat, A. (2022) 'Cervical cancer elimination in Indian context: Moving from barriers to facilitators', *Cancer*, 128(23), pp. 4041–4046. Available at: https://doi.org/10.1002/cncr.34486.
- Vu, M. *et al.* (2018) 'Cervical cancer worldwide', *Current Problems in Cancer*, 42(5), pp. 457–465. Available at: https://doi.org/10.1016/j.currproblcancer.2018.06.003.
- World Health Organization (n.d.) (WHO, n.d.) *Atlas of cytopathology*. International Agency for Research on Cancer. Available at: https://screening.iarc.fr/atlascyto.php (Accessed: 14 October 2024).

- Wilailak, S., Kengsakul, M. and Kehoe, S. (2021) 'Worldwide initiatives to eliminate cervical cancer', *International Journal of Gynaecology and Obstetrics: The Official Organ of the International Federation of Gynaecology and Obstetrics*, 155 Suppl 1(Suppl 1), pp. 102–106. Available at: https://doi.org/10.1002/ijgo.13879.
- Williamson, A.-L. (2023) 'Recent Developments in Human Papillomavirus (HPV) Vaccinology', *Viruses*, 15(7), p. 1440. Available at: https://doi.org/10.3390/v15071440.
- Wingo, S.N. *et al.* (2009) 'Somatic LKB1 mutations promote cervical cancer progression', *PloS One*, 4(4), p. e5137. Available at: https://doi.org/10.1371/journal.pone.0005137.
- Xue, P., Ng, M.T.A. and Qiao, Y. (2020) 'The challenges of colposcopy for cervical cancer screening in LMICs and solutions by artificial intelligence', *BMC medicine*, 18(1), p. 169. Available at: https://doi.org/10.1186/s12916-020-01613-x.
- Xue, W. *et al.* (2024) 'Wnt/β-catenin-driven EMT regulation in human cancers', *Cellular and molecular life sciences: CMLS*, 81(1), p. 79. Available at: https://doi.org/10.1007/s00018-023-05099-7.
- Yang, M. *et al.* (2018) 'Wnt signaling in cervical cancer?', *Journal of Cancer*, 9(7), pp. 1277–1286. Available at: https://doi.org/10.7150/jca.22005.
- Zeng, Q. *et al.* (2017) 'LKB1 inhibits HPV-associated cancer progression by targeting cellular metabolism', *Oncogene*, 36(9), pp. 1245–1255. Available at: https://doi.org/10.1038/onc.2016.290.
- Zhang, Y. *et al.* (2017) 'Direct HPV E6/Myc interactions induce histone modifications, Pol II phosphorylation, and hTERT promoter activation', *Oncotarget*, 8(56), pp. 96323–96339. Available at: https://doi.org/10.18632/oncotarget.22036.
- Zhong, Z. *et al.* (2012) '[Promoter methylation of DAPK1, RAR-β and MGMT in exfoliated cervical cytology and its clinical application]', *Zhonghua Fu Chan Ke Za Zhi*, 47(3), pp. 196–200.

S.no	NAME	AGE	PARITY	Parity (Nullipara vs Unipara vs Multipara)	UHID	LMP	MENSTURAL HISTORY
1	Ranju	50	P4L4	Multipara	239212300887626	15/04/2023	REGULAR
2	MEENA BAI	28	P2L2	Multipara	239212301130337	10/05/2023	REGULAR
3	ARUNA	38	P3L3	Multipara	239212200151282	26/05/2023	REGULAR
4	JYOTI	45	P3L3	Multipara	239212202324265	05/04/2023	REGULAR
5	KIRAN	52	P2L2	Multipara	239212301163117	2 YEAR BACK	POSTMENOPAUSAL
6	JIJIYA BAI	52	P4L4	Multipara	239212301260813	6 MONTH BACK	POSTMENOPAUSAL
7	MEENA BAI	73	P3L3	Multipara	239212300993876	15 years back	POSTMENOPAUSl
8	SHETA SHUKLA	58	P3L3	Multipara	239212301265203	5 YEAR BACK	POSTMENOPAUSAL
9	ANITA SAHU	38	P1L1	Unipara	239212301002601	06/04/2023	REGULAR
10	SHREMATIDEVI	55	P5L5	Multipara	239212301484479	2 YEARS BACK	POSTMENOPAUSAL
11	SHOBHA	43	P4L4	Multipara	239212301926986	25/08/2023	REGULAR
12	RAJANI	39	P5L3	Multipara	239212200360442	01/08/2023	REGULAR
13	NEETU	42	P2L2	Multipara	239212201806053	22/06/2023	REGULAR
14	DROPTI	50	P2L2	Multipara	239212301149394	01/06/2023	REGULAR
15	ANJU SONI45	44	P3L3	Multipara	239212300889921	09/06/2023	REGULAR
16	SANTOSH BAI	24	P2L2	Multipara	239212301352564	08/06/2023	REGULAR
17	TARA BAI	47	P2L2	Multipara	239212202594220	23/01/2023	REGULAR
18	SADHANA	41	P2L2	Multipara	239212300630528	05/03/2023	REGULAR
19	RAJANIBAI	39	P5L5	Multipara	239212200360442	23/05/2023	REGULAR
20	PRITI	38	P2L2	Multipara	239212300339777	16/04/2023	REGULAR
21	ASHADEVI	42	P2L2	Multipara	239212300957292	08/05/2023	REGULAR
22	BABITA	37	P2L2	Multipara	239212300934721	15/04/2023	REGULAR
23	SHOBHA	25	P1L1	Unipara	239212300740960	29/03/2023	REGULAR
24	LALITHA	41	P2L2	Multipara	239212202122697	07/05/2023	REGULAR
25	ARCHANA37	37	P2L2	Multipara	239212300810208	15/05/2023	REGULAR
26	SUDHA	52	P3L3	Multipara	239212300953203	1 YEAR BACK	POSTMENOPAUSAL
27	TRIVENI	55	P3L3	Multipara	23921230003312	2 YEARS BACK	POSTMENOPAUSAL
28	NEELAM	37	P2L2	Multipara	239212200043374	01/05/2023	REGULAR
29	RATABAI	39	P3L3	Multipara	23921230089903	02/05/2023	REGULAR
30	PINKY	41	P3L3	Multipara	236212300763811	05/05/2023	REGULAR
31	BASANTI	61	P5L5	Multipara	239212300667014	12 YEARS BACK	POSTMENOPAUSAL
32	REENA	28	P2L2	Multipara	239212300754642	28/03/2023	REGULAR
33	SUNITA	39	P2L2	Multipara	239212202576612	01/04/2023	REGULAR
34	RAJAL	32	P2L2	Multipara	239212300241246	05/04/2023	REGULAR
35	ANNAPURNA	40	P3L3	Multipara	239212300737772	21/03/2023	REGULAR
36	SANDHYA YADAV	40	P1L1	Unipara	239212300816371	20/03/2023	REGULAR
37	BABITA	33	P2L2	Multipara	239212300706478	07/03/2024	REGULAR
38	MAYA DEVI	60	P3L3	Multipara	239212200323971	10YEAR BACK	POSTMENOPAUSAL
39	GEETABAI	62	P4L4	Multipara	239212301988701	10YEAR BACK	POSTMENOPAUSAL
40	MEENA BAI	48	P2L2	Multipara	239212301920023	04/09/2023	REGULAR

41	SARITA	34	P2L2	Multipara	239212302039151	23/08/2023	REGULAR
42	VIJAYBAI	39	P3L3	Multipara	239212301706536	01/08/2023	REGULAR
43	LAXMI	50	P3L3	Multipara	239212301628551	14/06/2023	REGULAR
44	PAVITRA	50	P1L1	Unipara	239212302250391	20/08/2023	REGULAR
45	SHANTI	53	P3L3	Multipara	239212302022851	01/09/2023	REGULAR
46	ANTIMA SINGH	35	P2L2	Multipara	239212301995571	23/08/2023	REGULAR
47	SANTARA	64	P2L2	Multipara	239212302493277	10YEAR BACK	POSTMENOPAUSAL
48	KIRTI	39	P3L3	Multipara	239212302199140	01/10/2023	REGULAR
49	SHEELA	38	P3L3	Multipara	239212300550834	01/10/2023	REGULAR
50	AARTI	28	P2L2	Multipara	239212301919327	12/09/2023	REGULAR
51	KALA	65	P2L2	Multipara	239212302405611	15 years back	POSTMENOPAUSAL
52	DEVKI	40	P2L2	Multipara	239212302481139	12/09/2023	REGULAR
53	GEETA	54	P4L4	Multipara	239212302535425	10YEAR BACK	POSTMENOPAUSAL
54	MAMTA	42	P4L4	Multipara	239212302084806	25/08/2023	REGULAR
55	DURGA	44	P1L1	Unipara	239212202328767	10/10/2023	REGULAR
56	SAVITRI	42	P2L2	Multipara	239212201139618	08/10/2023	REGULAR
57	RAJKUMARI	48	P3L3	Multipara	239212302479274	22/08/2023	REGULAR
58	SUREKHA	37	P3L3	Multipara	239212302647029	20/10/2023	REGULAR
59	NOOR JAHAN	46	P2L2	Multipara	################	01/10/2023	REGULAR
60	HEMBAI	50	P4L4	Multipara	239212300789748	20/10/2023	REGULAR
61	MAMTA	42	P4L4	Multipara	239212302662648	02/10/2023	REGULAR
62	REKHADEVI	44	P3L3	Multipara	239212302599750	09/10/2023	REGULAR
63	MAMTA	43	P2L2	Multipara	239212302637651	15/10/2023	REGULAR
64	SANGEETA	40	P2L2	Multipara	239212202420570	29/10/2023	REGULAR
65	0MBATI	47	P4L4	Multipara	239212302692385	20/10/2023	REGULAR
66	SHILU	47	P2L2	Multipara	239212301403533	10/10/2023	REGULAR
67	SHRADDHA	26	P0L0	Nullipara	239212302551633	09/10/2023	REGULAR
68	AARTI	34	P1L1	Unipara	239212302185157	28/10/2023	REGULAR
69	LATA	44	P1L1	Unipara	239212302600685	23/10/2023	REGULAR
70	ARUNA GUPTA	48	P4L4	Multipara	239212302720559	28/09/2023	REGULAR
71	LAXMIBAI	48	P3L3	Multipara	239212302189659	20/08/2023	REGULAR
72	RAJKUMARI	48	P3L3	Multipara	239212302762499	10/11/2023	REGULAR
73	RASHMI	46	P3L3	Multipara	239212302658071	24/10/2023	REGULAR
74	SHIVWATI	52	P3L3	Multipara	239212302780357	3 MONTH BACK	REGULAR
75	MADHU	49	P3L3	Multipara	239212201863987	15/11/2023	REGULAR
76	MAYA	46	P3L3	Multipara	239212302898056	10/11/2023	REGULAR
77	HEMLATA	43	P5L5	Multipara	239212302849349	14/11/2023	REGULAR
78	POOJA	37	P2L2	Multipara	239212302826811	24/11/2023	REGULAR
79	GITA SONI	47	P3L3	Multipara	239212302623405	28/11/2023	REGULAR
80	KANTI	47	P2L2	Multipara	239212302902061	20/11/2023	REGULAR
81	RAMA	37	P3L3	Multipara	239212303013883	12/12/2023	REGULAR
82	MITELESH	44	P2L2	Multipara	239212303027628	05/12/2023	REGULAR
83	RAMVATI	55	P4L4	Multipara	239212302835437	07/12/2023	REGULAR
84	MAYA YADAV	49	P4L4	Multipara	239212303073001	07/12/2023	REGULAR
85	SUMAN	47	P3L3	Multipara	239212201915545	10/11/2023	REGULAR
86	MAYA SHUKLA	40	P2L2	Multipara	239212401991424	10/08/2024	REGULAR

87	PHOOLWATI	61	P5L5	Multipara	239212401827870	12 YEARS BACK	POSTMENOPAUSAL
88	SARASWATI	47	P2L2	Multipara	239212401981267	21/07/2024	REGULAR
89	SUNITA	44	P3L3	Multipara	239212300815293	14/07/2024	REGULAR
90	PRAVESHBAI	40	P3L3	Multipara	239212401977006	07/07/2024	REGULAR
91	PHOOLMATI	52	P4L4	Multipara	239212402153457	5 YEAR BACK	PM
92	PREETI	35	P3L3	Multipara	239212402079087	07/07/2024	REGULAR
93	KUSUM	42	P3L3	Multipara	239212300451861	31/07/2024	REGULAR
94	UMRANI	48	P2L2	Multipara	239212401689833	30/06/2024	REGULAR
95	SARIKA	41	P3L3	Multipara	239212400133742	03/06/2024	REGULAR
96	KAMLESH	57	P2L2	Multipara	239212300276091	6 YEAR BACK	PM
97	LEELABAI	51	P4L4	Multipara	239212401790682	5 YEAR BACK	PM
98	REKHA	44	P3L3	Multipara	239212401814361	18/07/2024	REGULAR
99	FUTRI	40	P1L1	Unipara	239212401818978	15/06/2024	REGULAR
100	SUKHWATI	50	P2L2	Multipara	239212101865488	29/06/2024	REGULAR
101	Ranju	50	P4L4	Multipara	239212300887626	15/04/2023	REGULAR
102	MEENA BAI	28	P2L2	Multipara	23921230113037	10/05/2023	REGULAR
103	ARUNA	38	P3L3	Multipara	239212200151282	26/05/2023	REGULAR
104	JYOTI	45	P3L3	Multipara	239212202324265	05/04/2023	REGULAR
105	KIRAN	52	P2L2	Multipara	239212301163117	2 YEAR BACK	POSTMENOPAUSAL
106	JIJIYA BAI	52	P4L4	Multipara	239212301260813	6 MONTH BACK	POSTMENOPAUSAL
107	MEENA BAI	73	P3L3	Multipara	239212300993876	15 years back	POSTMENOPAUSl
108	SHETA SHUKLA	58	P3L3	Multipara	239212301265203	5 YEAR BACK	POSTMENOPAUSAL
109	ANITA SAHU	38	P1L1	Unipara	239212301002601	06/04/2023	REGULAR
110	SHREMATIDEVI	55	P5L5	Multipara	239212301484479	2 YEARS BACK	POSTMENOPAUSAL
111	SHOBHA	43	P4L4	Multipara	239212301926986	25/08/2023	REGULAR
112	RAJANI	39	P5L3	Multipara	239212200360442	01/08/2023	REGULAR
113	NEETU	42	P2L2	Multipara	239212201806053	22/06/2023	REGULAR
114	DROPTI	50	P2L2	Multipara	239212301149394	01/06/2023	REGULAR
115	ANJU SONI45		P3L3	Multipara	239212300889921	09/06/2023	REGULAR
116	SANTOSH BAI	24	P2L2	Multipara	239212301352564	08/06/2023	REGULAR
117	TARA BAI	47	P2L2	Multipara	239212202594220	23/01/2023	REGULAR
118	SADHANA	41	P2L2	Multipara	239212300630528	05/03/2023	REGULAR
119	RAJANIBAI	39	P5L5	Multipara	239212200360442	23/05/2023	REGULAR
120	PRITI	38	P2L2	Multipara	239212300339777	16/04/2023	REGULAR
121	ASHADEVI	42	P2L2	Multipara	239212300957292	08/05/2023	REGULAR
122	BABITA	37	P2L2	Multipara	239212300934721	15/04/2023	REGULAR
123	SHOBHA	25	P1L1	Unipara	239212300740960	29/03/2023	REGULAR
124	LALITHA	41	P2L2	Multipara	239212202122697	07/05/2023	REGULAR
125	ARCHANA37	37	P2L2	Multipara	239212300810208	15/05/2023	REGULAR
126	SUDHA	52	P3L3	Multipara	239212300953203	1 YEAR BACK	POSTMENOPAUSAL
127	TRIVENI	55	P3L3	Multipara	23921230003312	2 YEARS BACK	POSTMENOPAUSAL
128	NEELAM	37	P2L2	Multipara	239212200043374	01/05/2023	REGULAR
129	RATABAI	39	P3L3	Multipara	23921230089903	02/05/2023	REGULAR
130	PINKY	41	P3L3	Multipara	236212300763811	05/05/2023	REGULAR
131	BASANTI	61	P5L5	Multipara	239212300667014	12 YEARS BACK	POSTMENOPAUSAL

132	REENA	28	P2L2	Multipara	239212300754642	28/03/2023	REGULAR
133	SUNITA	39	P2L2	Multipara	239212202576612	01/04/2023	REGULAR
134	RAJAL	32	P2L2	Multipara	239212300241246	05/04/2023	REGULAR
135	ANNAPURNA	40	P3L3	Multipara	239212300737772	21/03/2023	REGULAR
136	SANDHYA YADAV	40	P1L1	Unipara	239212300816371	20/03/2023	REGULAR
137	BABITA	33	P2L2	Multipara	239212300706478	07/03/2024	REGULAR
138	MAYA DEVI	60	P3L3	Multipara	239212200323971	10YEAR BACK	POSTMENOPAUSAL
139	GEETABAI	62	P4L4	Multipara	239212301988701	10YEAR BACK	POSTMENOPAUSAL
140	MEENA BAI	48	P2L2	Multipara	239212301920023	04/09/2023	REGULAR
141	SARITA	34	P2L2	Multipara	239212302039151	23/08/2023	REGULAR
142	VIJAYBAI	39	P3L3	Multipara	239212301706536	01/08/2023	REGULAR
143	LAXMI	50	P3L3	Multipara	239212301628551	14/06/2023	REGULAR
144	PAVITRA	50	P1L1	Unipara	239212302250391	20/08/2023	REGULAR
145	SHANTI	53	P3L3	Multipara	239212302022851	01/09/2023	REGULAR
146	ANTIMA SINGH	35	P2L2	Multipara	239212301995571	23/08/2023	REGULAR
147	SANTARA	64	P2L2	Multipara	239212302493277	10YEAR BACK	POSTMENOPAUSAL
148	KIRTI	39	P3L3	Multipara	239212302199140	01/10/2023	REGULAR
149	SHEELA	38	P3L3	Multipara	239212300550834	01/10/2023	REGULAR
150	RUBI BAI	30	P2L2	Multipara	239212302680905	12/09/2023	REGULAR
151	VARSHA	30	P2L2	Multipara	239212302752876	16/11/2023	REGULAR
152	SUSHILA NAGAR	32	P2L2	Multipara	239212100123001	05/10/2023	REGULAR
153	NIRAJ SINHA	55	P2L2	Multipara	239212302953197	28/10/2023	REGULAR
154	NISHA YADAV	61	P2L2	Multipara	23921201895711	10 YEARS BACK	POST MENOPAUSAL
155	SAROJ PATEL	40	P2L2	Multipara	239212302900778	02/12/2023	REGULAR
156	GEETA BAI	62	P4L4	Multipara	239212301988701	12 YEARS BACK	POST MENOPAUSAL
157	ARUNA TRIPATHI	53	P2L2	Multipara	239212302999910	4 YEARS BACK	POST MENOPAUSAL
158	SIYA	63	P2L2	Multipara	239212303022413	14 YEARS BACK	POST MENOPAUSAL
159	TARA BAI	45	P2L2	Multipara	239212202439721	28/12/2023	REGULAR
160	BINA RAMA	60	P3L3	Multipara	239212302013888	11 YEARS BACK	REGULAR
161	MAHASWETA DEY	36	P1L1	Unipara	239212302853095	24/12/2023	REGULAR
162	ASHA SAHU	46	P3L3	Multipara	239212400056144	01/01/2024	IRREGULAR
163	HEMLATA	44	P5L5	Multipara	239212302849349	28/12/2023	REGULAR
164	SUNITA LOWANSHI	39	P2L1	Multipara	239212400073359	09/01/2024	REGULAR
165	ASHA PATEL	40	P4L4	Multipara	239212202528320	12/01/2024	REGULAR
166	MAMTA BAI	40	P2L2	Multipara	239212201353121	12/01/2024	REGULAR
167	SAVITRI	63	P5L5	Multipara	239212400222672	14 YEARS BACK	REGULAR
168	VISHNA DEVI	37	P2L2	Multipara	239212300214109	27/12/2023	REGULAR
169	KAMAL MALVIYA	48	P3L3	Multipara	239212400222991	25/01/2024	REGULAR
170	NIRMALA	45	P4L4	Multipara	239212201955814	26/01/2024	REGULAR
171	SUMITRA BAI	68	P3L3	Multipara	239212400232899	19 YEARS BACK	POST MENOPAUSAL
172	AARTI AHIRWAR	22	P4L4	Multipara	239212400300339	01/02/2024	REGULAR
173	SHEELA AHIRWAR	47	P4L4	Multipara	239212400254779	06/11/2023	REGULAR

174	SUNITA SANDHIYHA	44	P3L3	Multipara	239212400468001	BLEEDING FROM 10 MONTHS CONTINUOUS	IRREGULAR
175	ANITA SAHU	48	P4L4	Multipara	239212301742583	06/10/2023	REGULAR
176	LALTA BAI	50	P4L4	Multipara	239212302230420	2 YEARS BACK	REGULAR
177	HEENA KUMARI	30	P0L0	Nullipara	239212302583658	01/10/2023	REGULAR
178	CHANDRAKALA PANDOLE	49	P4L4	Multipara	239212100127910	14/10/2023	REGULAR
179	RAMKALI BAI	35	P4L4	Multipara	239212302153701	06/11/2023	REGULAR
180	GULAB BAI	58	P3L3	Multipara	239212301965752	18 YEARS BACK	POST MENOPAUSAL
181	SARJU BAI	43	P4L4	Multipara	239212302200091	03/11/2023	REGULAR
182	KUSMA PAL	40	P4L4	Multipara	239212301469682	03/11/2023	REGULAR
183	DURGESHWARI	45	P3L3	Multipara	239212200763792	07/11/2023	REGULAR
184	SHIKHA	25	P0L0	Nullipara	239212302180601	25/08/2023	REGULAR
185	NIRMALA	41	P2L2	Multipara	239212302544009	29/09/2023	REGULAR
186	BHURI	50	P2L2	Multipara	239212301984005	5 YEARS BACK	POSTMENOPAUSAL
187	SUMITRA 46		P2L2	Multipara	239212301301935	23/07/2023	REGULAR
188	RADHA	46	P2L2	Multipara	239212301967844	11/08/2024	REGULAR
189	VINITA	52	P2L2	Multipara	239212201181135	28/07/2024	REGULAR
190	MANJU	47	P1L1	Unipara	239212200268244	22/08/2023	REGULAR
191	SEETA	47	P5L5	Multipara	239212301847342	14/08/2023	REGULAR
192	KRISHNA	45	P5L5	Multipara	239212301927753	15/08/2023	REGULAR
193	KAMLESH	38	P3L3	Multipara	239212301620534	20/08/2023	REGULAR
194	BISSOBAI	47	P4L4	Multipara	239212302041287	6 MONTH BACK	PERIMENOPAUSAL
195	MANJU JAIN	48	P3L3	Multipara	239212302115167	25/07/2023	IRREGULAR
196	SHANTIBAI	47	P3L3	Multipara	239212302080703	20/08/2023	IRREGULAR
197	UJAWALA	50	P2L2	Multipara	239212302118531	23/08/2023	REGULAR
198	USHA	48	P2L2	Multipara	239212302093465	26/08/2023	REGULAR
199	ARCHANA37	39	P2L2	Multipara	239212301501845	19/09/2023	REGULAR
200	GAURA BAI	48	P3L3	Multipara	239212200660454	25/09/2023	REGULAR
201	Sandhna Raghuwanshi	41	P2L2	Multipara	239212300630528	01/03/2023	REGULAR
202	Raja Bai	38	P2L2	Multipara	239212201558823	09/02/2024	REGULAR
203	Ram Bai	40	P2L2	Multipara	239212202349667	16/02/2023	IRREGULAR
204	Purnima Kushwaha	46	P2L2	Multipara	239212300659836	09/02/2024	IRREGULAR
205	Guddi	45	P4L4	Multipara	239212300826651	16/02/2023	REGULAR
206	Ansu Bai	47	P3L3	Multipara	239212300742482	24/03/2023	REGULAR
207	Koshalya Kheer	44	P2L2	Multipara	239212300562727	11/04/2024	REGULAR
208	Kamla Bai	55	P3L3	Multipara	239212300733891	Postmenopausal	REGULAR
209	Kaliya Bai	85	P6L6	Multipara	239212300813771	Postmenopausal	REGULAR
210	Sarita Bachle	44	P0L0	Nullipara	239212300828476	Mensturating	REGULAR
211	Nirmala Bai	50	P2L2	Multipara	239212300765555	17/04/2023	REGULAR
212	Rajkumari Vishwakarma	42	P2L2	Multipara	239212300890660	Mensturating	REGULAR
213	Sudha Nagle	45	P2L2	Multipara	239212300833290	18/04/2023	REGULAR
214	Urmila	64	P4L4	Multipara	239212300660443	Postmenopausal	REGULAR
215	Maya Devi	49	P4L4	Multipara	239212200437186	07/08/2022	REGULAR
216	Shakila Khan	62	P0L0	Nullipara	239212201579227	Postmenopausal	REGULAR

217	Vinita Bai	34	P3L2D1	Multipara	239212300844404	21/04/2023	REGULAR
218	Anshu P	38	P0L0	Nullipara	239212300950549	23/04/2023	REGULAR
219	Meera	38	P2L2	Multipara	239212300706745	04/05/2023	REGULAR
220	Ramdulari	31	P3L3	Multipara	239212300886824	15/04/2023	IRREGULAR
221	Deepika Joshi	34	P2L2	Multipara	239212201798026	Mensturating	REGULAR
222	Prabha Jain	38	P3L3	Multipara	239212202484017	23/04/2023	REGULAR
223	Leela Sahu	48	P2L2	Multipara	239212300958574	20/04/2024	REGULAR
224	Nisha Bai	36	P2L2	Multipara	239212300817431	01/05/2023	REGULAR
225	Geeta	60	P2L2	Multipara	239212201418461	Postmenopausal	REGULAR
226	Manju Shrivastav	45	P2L2	Multipara	239212301009771	Postmenopausal	REGULAR
227	Shushila Bai Patel	53	P4L4	Multipara	239212100124555	30/04/2023	IRREGULAR
228	Rajo Bai	48	P2L2	Multipara	239212300923789	27/04/2023	REGULAR
229	Sujata Prajapati	33	P1L1	Unipara	239212202233979	25/04/2023	REGULAR
230	Preet Chouhan	43	P2L2	Multipara	239212202188248	23/04/2023	REGULAR
231	Pushpa Devi	66	P5L5	Multipara	239212100145277	Postmenopausal	REGULAR
232	Salma Khan	42	P0L0	Nullipara	239212300482626	30/04/2023	REGULAR
233	Bharti Rai	43	P2L2	Multipara	239212301322740	20/04/2023	IRREGULAR
234	Shobha	25	P0L0	Nullipara	239212300740960	29/03/2023	REGULAR
235	Shanti Bai	32	P2L2	Multipara	239212300537684	26/03/2023	IRREGULAR
236	Prabha Devi	45	P3L3	Multipara	239212301002377	25/04/2023	REGULAR
237	Giriraj Katare	42	P2L2	Multipara	239212301111354	15/02/2023	IRREGULAR
238	Madhubala	38	P1L1	Unipara	239212301123191	26/04/2023	REGULAR
239	Anamika Rai	45	P2L2	Multipara	239212200792466	24/05/2023	REGULAR
240	Aarti Nigam	48	P2L2	Multipara	239212300789616	20/05/2023	REGULAR
241	Pankaja Jain	51	P3L3	Multipara	239212100031166	05/05/2023	REGULAR
242	Saraswati	55	P3L3	Multipara	239212300825469	Postmenopausal	REGULAR
243	Ram Kumari	38	P2L2	Multipara	239212301098927	22/05/2023	REGULAR
244	Poonam Sisodiya	40	P3L3	Multipara	239212300365514	07/05/2023	REGULAR
245	Komal Lulla	50	P2L2	Multipara	239212202373487	Postmenopausal	REGULAR
246	Gayatri Bai	48	P3L3	Multipara	239212300985571	31/05/2023	REGULAR
247	Savitri	45	P3L3	Multipara	239212100064439	29/05/2023	REGULAR
248	Anusuiya Ghorpade	50	P2L2	Multipara	239212301236564	04/06/2023	IRREGULAR
249	Kshama	24	P0L0	Nullipara	239212201610248	20/05/2023	REGULAR
250	Satyabhama Bai	44	P2L2	Multipara	239212301260538	01/07/2023	REGULAR
251	Vinita Singh	45	P2L2	Multipara	239212301194951	22/05/2023	REGULAR
252	Maya	40	P3L3	Multipara	239212301286022	21/05/2023	REGULAR
253	Ramkali	44	P3L3	Multipara	239212300983374	13/06/2023	REGULAR
254	Anita Jain	51	P3L3	Multipara	239212200515110	10/06/2023	REGULAR
255	Shanti Bai	36	P2L2	Multipara	239212201087561	Post Vaginal Hysterectomy	REGULAR
256	Sapna Wadhwaw	47	P2L2	Multipara	239212301328057	08/05/2023	REGULAR
257	Sumantra Parmar	38	P2L2	Multipara	239212300631630	20/05/2023	REGULAR
258	Chandra rekha	48	P3L3	Multipara	239212301376137	08/06/2024	REGULAR
259	Mousmi Khilar	38	P1L1	Unipara	239212200126911	10/06/2023	REGULAR
260	Janki Patel	46	P4L4	Multipara	239212301279654	01/06/2023	REGULAR
261	Kankshi Khare	39	P2L2	Multipara	239212301344952	04/06/2023	REGULAR
262	Aasha Bai	48	P3L3	Multipara	239212301341198	11/06/2023	REGULAR

263	Ram Devi	57	P4L4	Multipara	239212301415591	09/06/2023	REGULAR
264	Swarupa Kumari	40	P2L2	Multipara	239212301278291	29/05/2023	REGULAR
265	Saraswati	48	P3L3	Multipara	239212300860621	D10 on menses	REGULAR
266	Reena Singh	40	P4L4	Multipara	239212301306392	07/06/2023	REGULAR
267	Tara Bushwaha	45	P2L2	Multipara	239212301334183	05/06/2023	REGULAR
268	Sushma Rani	47	P2L2	Multipara	239212201845261	31/05/2023	REGULAR
269	Pinky Jain	41	P2L3	Multipara	239212301203135	27/06/2023	REGULAR
270	Savitri Shukla	55	P3L3	Multipara	239212301378946	24/06/2023	REGULAR
271	Foolvati Bai	52	P4L4	Multipara	239212301256492	26/06/2023	REGULAR
272	Soniya Tekam	30	P2L2	Multipara	239212202051731	26/06/2023	REGULAR
273	Leela	35	P2L3	Multipara	239212301480180	28/06/2023	REGULAR
274	Hemvati Bai	55	P0L0	Nullipara	239212301518021	06/07/2023	REGULAR
275	Radhika Yadav	58	P2L2	Multipara	239212300415775	Postmenopausal	REGULAR
276	Usui Shahbad Puri	45	P2L2	Multipara	239212301531795	04/07/2023	REGULAR
277	Varsha	43	P3L2	Multipara	239212301508351	06/07/2023	REGULAR
278	Choti Bai	37	P3L3	Multipara	239212100025905	10/06/2023	REGULAR
279	Lalita	40	P2L2	Multipara	23921220232106	09/07/2023	REGULAR
280	Saroj Rani	54	P4L4	Multipara	239212301533844	10/07/2023	REGULAR
281	Saroj Malviya	45	P2L2	Multipara	239212202115127	Postmenopausal	REGULAR
282	Usha Mishra	47	P3L3	Multipara	239212200241427	07/07/2023	REGULAR
283	Lalita Jhanbade	45	P3L4	Multipara	239212300478947	02/06/2023	REGULAR
284	Asha Bai	54	P4L4	Multipara	239212301608810	14/07/2023	REGULAR
285	Seema Singh	39	P2L2	Multipara	239212201804204	06/07/2023	REGULAR
286	Kiran Bai Soni	50	P3L3	Multipara	239212301709063	08/07/2023	REGULAR
287	Rama Bai	33	P3L3	Multipara	239212301414888	20/07/2023	REGULAR
288	Bhagwati Bai	46	P3L3	Multipara	239212301044061	26/07/2023	REGULAR
289	Sarika	48	P2L2	Multipara	239212301713664	12/07/2023	REGULAR
290	Sanchita	45	P0L0	Nullipara	239212301780833	13/07/2023	REGULAR
291	Sanju Devi	29	P2L2	Multipara	239212301253779	20/05/2023	REGULAR
292	Chandrakala	49	P4L4	Multipara	239212100127970	03/08/2023	IRREGULAR
293	Suman Bai	45	P6L6	Multipara	239212301398084	04/07/2023	REGULAR
294	Sushma Dubey	48	P4L4	Multipara	239212301721586	Postmenopausal	REGULAR
295	Ramakanti	40	P2L2	Multipara	239212301581202	25/07/2023	REGULAR
296	Nirmala Sharma	49	P2L2	Multipara	239212200463578	Postmenopausal	REGULAR
297	Gyarsi Pal	58	P3L3	Multipara	239212301705203	11/08/2023	REGULAR
298	Savita Nagar	39	P2L2	Multipara	239212301846621	12/07/2023	IRREGULAR
299	Hemlata Yadav	59	P3L3	Multipara	239212301888014	Postmenopausal	REGULAR
300	Kala Bai Sahu	60	P4L4	Multipara	239212200197860	Postmenopausal	REGULAR

S.no	USE OF CONTRACEPTION	PRESENTING COMPLAIN	MEDICAL OR SURGICAL HISTORY	ANY H/O SEXUALLY TRANSMITTED DISEASE	FAMILY HISTORY OF CERVICAL CANCER
1	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
2	NO	ABDOMEN DISTENTION,ABDO PAN	NO	NO	NO

3	NO	WHITE DISCHARGE	NO	NO	NO
4	NO	DYSMENORRHEA,DYSPAREUNIA	NO	NO	NO
5	NO	LOWER PAIN ABDO	NO	NO	NO
6	NO	POATMENOPAUSAL BLEEDING	NO	NO	NO
7	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
8	NO	POST MENOPAUSAL BLEEDING	NO	NO	NO
9	NO	SEVERE DYSMENORRHEA,WHITE DISCHARGE	NO	NO	NO
10	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
11	NO	PROLONGED MENSTURAL BLEEDING	TUBAL LIGATED	NO	NO
12	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
13	NO	WHITE DISCHARGE	NO	NO	NO
14	NO	LOWER ABDO PAIN	NO	NO	NO
15	NO	WHITE DISCHARGE	TUBAL LIGATED	NO	NO
16	NO	WHITE DISCHARGE	NO	NO	NO
17	NO	WHITE DISCHARGE	NO	NO	NO
18	NO	PAIN LOWER ABDO	NO	NO	NO
19	NO	POST COITAL BLEEDING	TUBAL LIGATED	NO	NO
20	NO	LOWER PAIN ABDO	NO	NO	NO
21	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
22	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
23	YES	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
24	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
25	YES	HEAVY MENSES	NO	NO	NO
26	YES	YELLOWISH DISCHARGE	NO	NO	NO
27	NO	WHITE DISCHARGE	NO	NO	NO
28	NO	WHITE DISCHARGE	NO	NO	NO
29	NO	WHITE DISCHARGE	NO	NO	NO
30	NO	SEVERE DYSMENORRHEA,WHITE DISCHARGE	NO	NO	NO
31	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
32	NO	WHITE DISCHARGE	NO	NO	NO
33	NO	WHITISH DISCHARGE	NO	NO	NO
34	NO	LOWER ABDO PAIN	NO	NO	NO
35	NO	LOWER ABDO PAIN	NO	NO	NO
36	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
37	NO	WHITISH DISCHARGE	NO	NO	NO
38	NO	WHITE DISCHARGE	NO	NO	NO
39	NO	PAIN LOWER ABDO	NO	NO	NO
40	NO	WHITE DISCHARGE	NO	NO	NO
41	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
42	NO	SEVERE DYSMENORRHEA,WHITE DISCHARGE	NO	NO	NO
43	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
44	NO	,WHITE DISCHARGE	NO	NO	NO
45	YES	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
46	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
47	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
48	NO	WHITE DISCHARG	NO	NO	NO

49	NO	WHITE DISCHARGE	NO	NO	NO
50	NO	LOWER ABDO PAIN	NO	NO	NO
51	NO	ABDOMEN DISTENTION,ABDO PAN	NO	NO	NO
52	NO	AUB -L	NO	NO	NO
53	NO	POSTMENOPAUSAL BLEEDING	NO	NO	NO
54	NO	HMB	NO	NO	NO
55	NO	HMB	NO	NO	NO
56	NO	HMB	NO	NO	NO
57	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
58	NO	HMB	NO	NO	NO
59	NO	AUB-A	NO	NO	NO
60	NO	HMB	NO	NO	NO
61	NO	AUB -L	NO	NO	NO
62	NO	AUB-P	NO	NO	NO
63	NO	HMB	NO	NO	NO
64	NO	HMB	NO	NO	NO
65	NO	HMB	NO	NO	NO
66	NO	AUB-L	NO	NO	NO
67	NO	AUB-L	NO	NO	NO
68	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
69	NO	HMB	NO	NO	NO
70	NO	AUB-A	NO	NO	NO
71	NO	WHITE DISCHARGE	NO	NO	NO
72	NO	POLYMENORRHEA	NO	NO	NO
73	NO	AUB-L	NO	NO	NO
74	NO	AUB-L	NO	NO	NO
75	NO	HMB	NO	NO	NO
76	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
77	NO	AUB-A	NO	NO	NO
78	NO	AUB-A	NO	NO	NO
79	NO	IRREGULAR MENSES	NO	NO	NO
80	NO	HMB	NO	NO	NO
81	NO	AUB-A	NO	NO	NO
82	NO	HMB	NO	NO	NO
83	NO	HMB	NO	NO	NO
84	NO	AUB-A	NO	NO	NO
85	NO	AUB -L	NO	NO	NO
86	NO	PID	NO	NO	NO
87	NO	ENDOMETRIAL POLYP	NO	NO	NO
88	NO	AUB-P	NO	NO	NO
89	NO	IRREGULAR MENSES	NO	NO	NO
90	NO	HMB	NO	NO	NO
91	NO	3 RD DEGREE UV PROLAPSE	NO	NO	NO
92	NO	AUB-A	NO	NO	NO
93	NO	AUB-L	NO	NO	NO
94	NO	POLYMENORRHEA	NO	NO	NO

95	NO	AUB	NO	NO	NO
96	NO	PMB	NO	NO	NO
97	NO	PMB	NO	NO	NO
98	NO	HMB	NO	NO	NO
99	NO	IRREGULAR MENSES	NO	NO	NO
100	NO	AUB -L	NO	NO	NO
101	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
102	NO	ABDOMEN DISTENTION,ABDO PAN	NO	NO	NO
103	NO	WHITE DISCHARGE	NO	NO	NO
104	NO	DYSMENORRHEA,DYSPAREUNIA	NO	NO	NO
105	NO	LOWER PAIN ABDO	NO	NO	NO
106	NO	POATMENOPAUSAL BLEEDING	NO	NO	NO
107	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
108	NO	POST MENOPAUSAL BLEEDING	NO	NO	NO
109	NO	SEVERE DYSMENORRHEA,WHITE DISCHARGE	NO	NO	NO
110	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
111	NO	PROLONGED MENSTURAL BLEEDING	TUBAL LIGATED	NO	NO
112	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
113	NO	WHITE DISCHARGE	NO	NO	NO
114	NO	LOWER ABDO PAIN	NO	NO	NO
115	NO	WHITE DISCHARGE	TUBAL LIGATED	NO	NO
116	NO	WHITE DISCHARGE	NO	NO	NO
117	NO	WHITE DISCHARGE	NO	NO	NO
118	NO	PAIN LOWER ABDO	NO	NO	NO
119	NO	POST COITAL BLEEDING	TUBAL LIGATED	NO	NO
120	NO	LOWER PAIN ABDO	NO	NO	NO
121	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
122	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
123	YES	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
124	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
125	YES	HEAVY MENSES	NO	NO	NO
126	YES	YELLOWISH DISCHARGE	NO	NO	NO
127	NO	WHITE DISCHARGE	NO	NO	NO
128	NO	WHITE DISCHARGE	NO	NO	NO
129	NO	WHITE DISCHARGE	NO	NO	NO
130	NO	SEVERE DYSMENORRHEA,WHITE DISCHARGE	NO	NO	NO
131	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
132	NO	WHITE DISCHARGE	NO	NO	NO
133	NO	WHITISH DISCHARGE	NO	NO	NO
134	NO	LOWER ABDO PAIN	NO	NO	NO
135	NO	LOWER ABDO PAIN	NO	NO	NO
136	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
137	NO	WHITISH DISCHARGE	NO	NO	NO
138	NO	WHITE DISCHARGE	NO	NO	NO
139	NO	PAIN LOWER ABDO	NO	NO	NO
140	NO	WHITE DISCHARGE	NO	NO	NO

141	NO	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
142	NO	SEVERE DYSMENORRHEA,WHITE DISCHARGE	NO	NO	NO
143	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
144	NO	,WHITE DISCHARGE	NO	NO	NO
145	YES	PROLONGED MENSTURAL BLEEDING	NO	NO	NO
146	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
147	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
148	NO	WHITE DISCHARG	NO	NO	NO
149	NO	WHITE DISCHARGE	NO	NO	NO
150	NO	POST COITAL BLEEDING	NO	NO	NO
151	NO	WHITE DISCHARGE PV	NO	NO	NO
152	NO	WHITE DISCHARGE PV	NO	NO	NO
153	NO	PROLONGED MENSTRUAL BLEEDING	NO	NO	NO
154	NO	POST COITAL BLEEDING	NO	NO	NO
155	NO	DISCHARGE P/V	NO	NO	NO
156	NO	PAIN ABDOMEN (LOWER)	NO	NO	NO
157	NO	WHITE DISCHARGE PV	NO	NO	NO
158	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
159	NO	PROLONGED MENSTRUAL BLEEDING	NO	NO	NO
160	NO	POST MENOPAUSAL BLEEDING	NO	NO	NO
161	NO	POST COITAL BLEEDING	NO	NO	NO
162	NO	IRREGULAR MENSES	NO	NO	NO
163	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
164	NO	WHITE DISCHARGE PV	NO	NO	NO
165	NO	POST COITAL BLEEDING	NO	NO	NO
166	NO	POST COITAL BLEEDING	NO	NO	NO
167	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
168	NO	POST COITAL BLEEDING	NO	NO	NO
169	NO	FREQUENT MENSES	NO	NO	NO
170	NO	WHITE DISCHARGE PV	NO	NO	NO
171	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
172	NO	WHITE DISCHARGE PV	NO	NO	NO
173	NO	SOMETHING COMING OUT OF VAGINA	NO	NO	NO
174	NO	PROLONGED MENSTRUAL BLEEDING + DYSMENORRHOEA	NO	NO	NO
175	NO	PAIN DURING MENSES	NO	NO	NO
176	NO	POST MENOPAUSAL BLEEDING	NO	NO	NO
177	NO	POST COITAL BLEEDING	NO	NO	NO
178	NO	PAIN ABDOMEN (LOWER)	NO	NO	NO
179	NO	WHITE DISCHARGE PV	NO	NO	NO
180	NO	POST MENOPAUSAL BLEEDING	NO	NO	NO
181	NO	WHITE DISCHARGE PV	NO	NO	NO
182	NO	PAIN DURING MENSES	NO	NO	NO
183	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
184	NO	POST COITAL BLEEDING	NO	NO	NO
185	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
186	NO	SPOTTING PV,WHITE DISCHARGE	NO	NO	NO

187	NO	HEAVY MENS.BLEEDING	NO	NO	NO
188	NO	POLYMENORRHAGIA	NO	NO	NO
189	NO	LOWER ABDO PAIN	NO	NO	NO
190	NO	MENORRHAGIA	NO	NO	NO
191	NO	AUB-L	NO	NO	NO
192	NO	HEAVY MENS.BLEEDING	NO	NO	NO
193	NO	AUB-L	NO	NO	NO
194	NO	HMB	NO	NO	NO
195	NO	IRREGULAR MENSES	NO	NO	NO
196	NO	3 RD DEGREE UV PROLAPSE	NO	NO	NO
197	NO	HMB	NO	NO	NO
198	NO	AUB-L	NO	NO	NO
199	NO	AUB-L	NO	NO	NO
200	NO	POLYMENORRHAGIA	NO	NO	NO
201	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
202	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
203	NO	IMB	NO	NO	NO
204	NO	IMB	NO	NO	NO
205	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
206	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
207	NO	ADENOMYOSIS	NO	NO	NO
208	NO	POST MENOPAUSAL BLEEDING	NO	NO	NO
209	NO	PEMPHIGUS VULGARIS	NO	NO	NO
210	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
211	NO	SUBMUCOUS FIBROID	NO	NO	NO
212	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
213	NO	AUB-L	NO	NO	NO
214	NO	3º UV Prolapse	NO	NO	NO
215	NO	AUB	NO	NO	NO
216	NO	IMB	NO	NO	NO
217	YES	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
218	YES	LOWER ABDOMINAL PAIN	NO	NO	NO
219	NO	AUB-E	NO	NO	NO
220	NO	Hypomenorrhoea	NO	NO	NO
221	NO	AUB-L	NO	NO	NO
222	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
223	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
224	YES	SPOTTING BEFORE MENSES	NO	NO	NO
225	NO	SWELLING IN UMBLICAL AREA	NO	NO	NO
226	NO	PAINFUL MICTURITION	NO	NO	NO
227	NO	IRREGULAR MENSES	NO	NO	NO
228	NO	AUB-A	NO	NO	NO
229	NO	AUB-L	NO	NO	NO
230	NO	MENORRHAGIA	NO	NO	NO
231	NO	Primary PAC	NO	NO	NO
232	NO	CERVICITIS	NO	NO	NO

233	NO	INTERMENSTRUAL BLEEDING	NO	NO	NO
234	NO	MISSED PERIOD	NO	NO	NO
235	NO	IRREGULAR MENSES	NO	NO	NO
236	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
237	NO	IMB	NO	NO	NO
238	NO	SECONDARY INFERTILITY	NO	NO	NO
239	NO	3º UV Prolapse	NO	NO	NO
240	NO	CERVICAL ELONGATION	NO	NO	NO
241	NO	AUB-A/L	NO	NO	NO
242	NO	SOMETHING DESCENDING PV	NO	NO	NO
243	YES	AUB-L	NO	NO	NO
244	NO	AUB	NO	NO	NO
245	NO	ET THICKNESS	NO	NO	NO
246	YES	HEAVY MENSTURAL BLEEDING	NO	NO	NO
247	YES	AUB-L	NO	NO	NO
248	NO	IMB	NO	NO	NO
249	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
250	NO	AUB	NO	NO	NO
251	NO	Prolonged Menses	NO	NO	NO
252	NO	AUB-L	NO	NO	NO
253	YES	UTERUS FIBROID	NO	NO	NO
254	NO	POLYMENORRHAGIA	NO	NO	NO
255	NO	CA. CERVIX	NO	NO	NO
256	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
257	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
258	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
259	NO	Copper-T in-situ	NO	NO	NO
260	NO	AUB-L	NO	NO	NO
261	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
262	NO	AUB	NO	NO	NO
263	NO	POSTMENOPAUSAL BLEEDING	NO	NO	NO
264	NO	WANT CONCEPTION	NO	NO	NO
265	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
266	NO	3º UV Prolapse	NO	NO	NO
267	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
268	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
269	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
270	NO	UTERINE FIBROID	NO	NO	NO
271	NO	AUB	NO	NO	NO
272	NO	AUB	NO	NO	NO
273	NO	AUB	NO	NO	NO
274	NO	INTRAMURAL FIBROIDS	NO	NO	NO
275	NO	Endocervical growth	NO	NO	NO
276	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
277	NO	AUB	NO	NO	NO
278	NO	AUB-A	NO	NO	NO

279	NO	AUB-L	NO	NO	NO
280	NO	ABDOMINAL PAIN	NO	NO	NO
281	NO	POSTMENOPAUSAL BLEEDING	NO	NO	NO
282	NO	HEAVY MENSTURAL BLEEDING	NO	NO	NO
283	NO	HEAVY MENSTRUAL BLEEDING	NO	NO	NO
284	NO	AUB-P	NO	NO	NO
285	NO	AUB-L	NO	NO	NO
286	NO	AUB with HTN	NO	NO	NO
287	NO	DEGENERATION FIBROID	NO	NO	NO
288	NO	AUB-A	NO	NO	NO
289	NO	AUB-L	NO	NO	NO
290	NO	HTN	NO	NO	NO
291	NO	WHITE DISCHARGE	NO	NO	NO
292	NO	IRREGULAR MENSES	NO	NO	NO
293	NO	AUB-L	NO	NO	NO
294	NO	3º UV Prolapse	NO	NO	NO
295	NO	AUB UNDER EVALUATION	NO	NO	NO
296	NO	CA. ENDOMETRIUM	NO	NO	NO
297	NO	AUB-L	NO	NO	NO
298	NO	IRREGULAR MENSES	NO	NO	NO
299	NO	UTERUS PROLAPSE	NO	NO	NO
300	NO	POST HYSTERECTOMY	NO	NO	NO

S.no	P/A	P/S	P/V
1	SOFT,NON-TENDER,OBESE	CERVIX IS BULKY , BLEEDING +	UTERUS 6-8 WEEKS
2	SOFT,NON-TENDER	CERVIX VAGINA NORMAL	UTERUS NORMAL
3	SOFT NON TENDER	CX-BULKY ,WHITISH DISCHARGE+	UTERUS NORMAL SIZE ,FREE FORNIX
4	SOFT NON TENDER	CX-HYPERTROPHIED,NEBOTHIAN CYST +	UTERUS 8-10WEEK SIZE
5	SOFT NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS NORMALSIZE
6	SOFT NON TENDER	CX HYPEREMIC ,NOT BLEED ON TOUCH	UTERUS 6-8 WEEKS
7	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL
8	SOFT NON TENDER	CX-HYPERMIC,VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
9	SOFT NON TENDER	,BLEEDING+ON GOING MENSES	UTERUS A/V ,NORMAL SIZE
10	SOFT NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS NORMAL SIZE ,FREE FORNIX
11	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
12	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
13	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
14	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
15	SOFT NON TENDER	,WHITE DISCHARGE+	UTERUS NORMAL SIZE ,FREE FORNIX
16	SOFT NON TENDER	,WHITE DISCHARGE+	UTERUS A/V ,NORMAL SIZE
17	SOFT NON TENDER	WHITE DISCHARHE	UTERUS ,VAGINA NORMAL
18	SOFT NON TENDER	CX -HYPERTROPHIED	UTERUS A/V ,NORMAL SIZE
19	SOFT NON TENDER	CX-HYPERMIC BLEED ON TOUCH	UTERUS A/V ,NORMAL SIZE
20	SOFT NON TENDER	CX HYPERTROPHIED,WHITE DISCHARGE	UTERUS A/V ,NORMAL SIZE

21	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS A/V ,NORMAL SIZE
22	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
23	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
24	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V,NORMAL
25	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS A/V ,NORMAL SIZE
26	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
27	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
28	SOFT NON TENDER	WHITE DISCHARHE	UTERUS 6-8 WEEKS
29	SOFT NON TENDER	HEAVY MENSTURAL FLOW	UTERUS 6-8 WEEKS
30	SOFT NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS A/V NORMAL
31	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS A/V NORMAL
32	SOFT NON TENDER	CX HYPERTROPHIED VAGINA NORMAL	UTERUS A/V NORMAL
33	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
34	SOFT NON TENDER	CX HYPERTROPHIED,WHITE DISCHARGE	UTERUS A/V NORMAL
35	SOFT NON TENDER	CX BULKY	UTERUS A/V NORMAL
36	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS NORMAL FORNIX FREE
37	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
38	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
39	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
40	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
41	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
42	SOFT NON TENDER	CX BULKY WHITE DISCHARGE	UTERUS A/V NORMAL SIZE
43	SOFT NON TENDER	CX BULKY VAGINA NORMAL	UTERUS A/V NORMAL SIZE
44	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
45	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
46	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
47	SOFT NON TENDER	CX BULKY VAGINA NORMAL	UTERUS A/V NORMAL SIZE
48	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V,NORMAL
49	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
50	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
51	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS NORMAL
52	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
53	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
54	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS 6-8 WEEKS
55	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
56	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
57	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
58	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
59	SOFT NON TENDER	CX BULKY	UTERUS 8-10WEEK SIZE
60	SOFT NON TENDER	CX BULKY	UTERUS 8-10WEEK SIZE
61	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
62	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS,VAGINA NORMAL
63	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
64	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
65	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
66	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE

67	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
68	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
69	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
70	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
71	SOFT NON TENDER	CX BULKY	UTERUS ,VAGINA NORMAL
72	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
73	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
74	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
75	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
76	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS,VAGINA NORMAL
77	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
78	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
79	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS NORMAL SIZE ,FREE FORNIX
80	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS NORMAL SIZE ,FREE FORNIX
81	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
82	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
83	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
84	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
85	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 10 WEEK
86	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
87	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
88	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
89	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS BULKY
90	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
91	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
92	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
93	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
94	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
95	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
96	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
97	SOFT NON TENDER	CX BULKY	UTERUS 6-8 WEEKS
98	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
99	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
100	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
101	SOFT,NON-TENDER,OBESE	CERVIX IS BULKY , BLEEDING +	UTERUS 6-8 WEEKS
102	SOFT,NON-TENDER	CERVIX VAGINA NORMAL	UTERUS NORMAL
103	SOFT NON TENDER	CX-BULKY ,WHITISH DISCHARGE+	UTERUS NORMAL SIZE ,FREE FORNIX
104	SOFT NON TENDER	CX-HYPERTROPHIED,NEBOTHIAN CYST +	UTERUS 8-10WEEK SIZE
105	SOFT NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS NORMALSIZE
106	SOFT NON TENDER	CX HYPEREMIC ,NOT BLEED ON TOUCH	UTERUS 6-8 WEEKS
107	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL
108	SOFT NON TENDER	CX-HYPERMIC,VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
109	SOFT NON TENDER	,BLEEDING+ON GOING MENSES	UTERUS A/V ,NORMAL SIZE
110	SOFT NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS NORMAL SIZE ,FREE FORNIX
111	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
112	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX

113	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
114	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
115	SOFT NON TENDER	,WHITE DISCHARGE+	UTERUS NORMAL SIZE ,FREE FORNIX
116	SOFT NON TENDER	,WHITE DISCHARGE+	UTERUS A/V ,NORMAL SIZE
117	SOFT NON TENDER	WHITE DISCHARHE	UTERUS ,VAGINA NORMAL
118	SOFT NON TENDER	CX -HYPERTROPHIED	UTERUS A/V ,NORMAL SIZE
119	SOFT NON TENDER	CX-HYPERMIC BLEED ON TOUCH	UTERUS A/V ,NORMAL SIZE
120	SOFT NON TENDER	CX HYPERTROPHIED,WHITE DISCHARGE	UTERUS A/V ,NORMAL SIZE
121	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS A/V ,NORMAL SIZE
122	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
123	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
124	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V,NORMAL
125	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS A/V ,NORMAL SIZE
126	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
127	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
128	SOFT NON TENDER	WHITE DISCHARHE	UTERUS 6-8 WEEKS
129	SOFT NON TENDER	HEAVY MENSTURAL FLOW	UTERUS 6-8 WEEKS
130	SOFT NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS A/V NORMAL
131	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS A/V NORMAL
132	SOFT NON TENDER	CX HYPERTROPHIED VAGINA NORMAL	UTERUS A/V NORMAL
133	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
134	SOFT NON TENDER	CX HYPERTROPHIED,WHITE DISCHARGE	UTERUS A/V NORMAL
135	SOFT NON TENDER	CX BULKY	UTERUS A/V NORMAL
136	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS NORMAL FORNIX FREE
137	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
138	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
139	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
140	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
141	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
142	SOFT NON TENDER	CX BULKY WHITE DISCHARGE	UTERUS A/V NORMAL SIZE
143	SOFT NON TENDER	CX BULKY VAGINA NORMAL	UTERUS A/V NORMAL SIZE
144	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
145	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
146	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
147	SOFT NON TENDER	CX BULKY VAGINA NORMAL	UTERUS A/V NORMAL SIZE
148	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A?V NORMAL SIZE
149	SOFT NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
150	SOFT, NON TENDER	CERVIX - HYPEREMIC, BLEEDING ON TOUCH VAGINA - NORMAL	NORMAL
151	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
152	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
153	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
154	SOFT, NON TENDER	CERVIX - HYPEREMIC, BLEEDING ON TOUCH VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
155	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
156	SOFT, NON TENDER	CERVIX - HYPERTROPHIED ; VAGINA - NORMAL ; WHITE DISCHARGE PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER

157	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
158	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
159	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
160	SOFT, NON TENDER	CERVIX - HYPEREMIC, BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
161	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEED ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
162	SOFT, NON TENDER	CERVIX - BULKING ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
163	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
164	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL WHITE DISCHARGE- PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
165	SOFT, NON TENDER	CERVIX - HYPEREMEIC ; BLEED ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
166	SOFT, NON TENDER	CERVIX - HYPEREMEIC ; BLEED ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
167	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
168	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEED ON TOUCH , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
169	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
170	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
171	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
172	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
173	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
174	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
175	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
176	SOFT, NON TENDER	CERVIX - HYPEREMIC , BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
177	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEEDS ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
178	SOFT, NON TENDER	CERVIX - HYPERTROPHIED ; VAGINA - NORMAL ; WHITE DISCHARGE PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
179	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
180	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
181	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
182	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; NO DISCHARGE	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
183	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
184	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEEDS ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
185	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	
186	SOFT NON TENDER	CERVIX BULKY,SPOTTING+	UTERUS 6-8 WEEKS
187	SOFT NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS NORMAL A/V ,B/L FREE FORNIX NON TRNDR
188	SOFT NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS BULKY,B/L FFNT
189	SOFT NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
190	SOFT NON TENDER	CERVIX,VAGINA NORMAL	UTERUS NORMAL B/L FFNT
191	SOFT NON TENDER	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS SIZE 6- 8 WEEK,B/L FFNT
192	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS BULKY,B/L FFNT
193	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS BULKY,B/L FFNT
194	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS BULKY,B/L FFNT

195	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL,B/L FFNT
196	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS NORMAL,B/L FFNT
197	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS BULKY,B/L FFNT
198	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
199	SOFT NON TENDER	CX,VAGINA NORMAL	UTERUS 8-10WEEK SIZE
200	SOFT NON TENDER	CX ,VAGINA NORMAL	UTERUS 6 -8 WEEK
201	SOFT, NON-TENDER, OBESE	CERVIX IS BULKY , BLEEDING +	UTERUS 6-8 WEEKS
202	SOFT, TENDER	CERVIX IS BULKY , BLEEDING +	UTERUS NORMAL SIZE, FREE FORNIX
203	SOFT, NON TENDER	CX-BULKY, WHITISH DISCHARGE+	UTERUS NORMAL SIZE, FREE FORNIX
204	SOFT, NON TENDER	CERVIX IS BULKY, BLEEDING +	UTERUS NORMAL SIZE, FREE FORNIX
205	SOFT, NON TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS NORMALSIZE
206	SOFT, NON TENDER	CX HYPEREMIC, NOT BLEED ON TOUCH	UTERUS NORMALSIZE
207	SOFT, NON TENDER	CX, VAGINA NORMAL	UTERUS NORMAL
208	SOFT, NON TENDER	CX-HYPERMIC, VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
209	SOFT, NON TENDER	CX-HYPERMIC, VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
210	SOFT, NON TENDER	BLEEDING+ON GOING MENSES	UTERUS NORMAL SIZE ,FREE FORNIX
211	SOFT, NON TENDER	CX, VAGINA NORMAL	UTERUS 6-8 WEEKS
212	SOFT, NON TENDER	CERVIX IS BULKY , BLEEDING +	UTERUS NORMAL SIZE ,FREE FORNIX
213	SOFT, NON TENDER	CX,VAGINA NORMAL	UTERUS 10-12 WEEKS
214	SOFT, NON TENDER	CX BULKY VAGINA NORMAL	UTERUS NORMAL SIZE ,FREE FORNIX
215	SOFT, NON TENDER	CERVIX IS BULKY, BLEEDING +	UTERUS NORMAL SIZE ,FREE FORNIX
216	SOFT, NON TENDER	CX HYPEREMIC, NOT BLEED ON TOUCH	UTERUS A/V ,NORMAL SIZE
217	SOFT, NON TENDER	CERVIX IS BULKY , BLEEDING +	UTERUS ,VAGINA NORMAL
218	SOFT, TENDER	CX -HYPERTROPHIED	UTERUS A/V ,NORMAL SIZE
219	SOFT, NON TENDER	CX-HYPERMIC BLEED ON TOUCH	UTERUS A/V ,NORMAL SIZE
220	SOFT, NON TENDER	CX HYPERTROPHIED, WHITE DISCHARGE	UTERUS A/V ,NORMAL SIZE
221	SOFT, NON TENDER	CX BULKY,VAGINA NORMAL NO DISCHARGE	UTERUS 6-8 WEEKS
222	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
223	SOFT, NON TENDER	CERVIX IS BULKY, BLEEDING +	UTERUS A/V ,NORMAL SIZE
224	SOFT, NON TENDER	CX-HYPERMIC, BLEED ON TOUCH	UTERUS A/V,NORMAL
225	TENDERNESS (+), SWELLING IN UMBLICAL AREA	CX BULKY,VAGINA NORMAL NO DISCGARGE	UTERUS A/V ,NORMAL SIZE
226	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V ,NORMAL SIZE
227	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
228	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
229	SOFT, NON TENDER	CX,VAGINA NORMAL	UTERUS 6-8 WEEKS
230	SOFT, TENDER	CX BULKY ,WHITE DISCHARGE	UTERUS A/V NORMAL
231	SOFT, NON TENDER	CX,VAGINA NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
232	SOFT, NON TENDER	CX HYPEREMIC, NOT BLEED ON TOUCH	UTERUS A/V NORMAL
233	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
234	SOFT, NON TENDER	CX HYPERTROPHIED,WHITE DISCHARGE	UTERUS A/V NORMAL
235	SOFT, NON TENDER	CX BULKY	UTERUS A/V NORMAL
236	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS NORMAL FORNIX FREE
237	SOFT, NON TENDER	CX HYPEREMIC, NOT BLEED ON TOUCH	UTERUS A/V NORMAL
238	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
239	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL

240	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
241	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
242	SOFT, NON TENDER	CX BULKY WHITE DISCHARGE	UTERUS A/V NORMAL SIZE
243	SOFT, NON TENDER	CX BULKY VAGINA NORMAL	UTERUS 6-8 WEEKS
244	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL
245	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
246	SOFT, NON TENDER	CX HYPEREMIC, NOT BLEED ON TOUCH	UTERUS A/V NORMAL SIZE
247	SOFT, NON TENDER	CX BULKY VAGINA NORMAL	UTERUS 6-8 WEEKS
248	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
249	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS A/V NORMAL SIZE
250	SOFT, NON TENDER	CERVIX - HYPEREMIC, BLEEDING ON TOUCH VAGINA - NORMAL	NORMAL
251	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
252	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
253	SOFT, TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
254	SOFT, NON TENDER	CERVIX - HYPEREMIC, BLEEDING ON TOUCH VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
255	SOFT, NON TENDER	CERVIX - HYPEREMIC, BLEEDING ON TOUCH VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
256	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
257	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
258	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
259	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
260	SOFT, NON TENDER	CERVIX - HYPEREMIC, BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
261	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEED ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
262	SOFT, NON TENDER	CERVIX - BULKING ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
263	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
264	SOFT, NON TENDER	CERVIX - BULKY VAGINA - NORMAL WHITE DISCHARGE- PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
265	SOFT, NON TENDER	CERVIX - HYPEREMEIC ; BLEED ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
266	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
267	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
268	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEED ON TOUCH , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
269	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
270	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
271	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
272	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
273	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
274	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
275	SOFT, NON TENDER	CERVIX , VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
276	SOFT, NON TENDER	CERVIX - HYPEREMIC , BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
277	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEEDS ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
278	SOFT, NON TENDER	CERVIX - HYPERTROPHIED ; VAGINA - NORMAL ; WHITE DISCHARGE PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER

279	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS 6-8 WEEKS
280	SOFT, TENDER	CERVIX - BULKY ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
281	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
282	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; NO DISCHARGE	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
283	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
284	SOFT, NON TENDER	CERVIX - HYPEREMIC , BLEEDS ON TOUCH ; VAGINA - NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
285	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
286	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
287	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
288	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
289	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
290	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
291	SOFT, NON TENDER	CERVIX, VAGINA - NORMAL ; WHITE DISCHARGE +	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
292	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
293	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
294	SOFT, NON TENDER	CX VAGINA NORMAL	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
295	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
296	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
297	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
298	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
299	SOFT, NON TENDER	CERVIX - BULKY ; VAGINA - NORMAL ; BLEEDING - PRESENT	UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER
300	SOFT, NON TENDER	CX VAGINA NORMAL; UTERUS - ANTEVERTED , NORMAL BILATERAL FREE FORNIX, NON TENDER	

S.no	PAP SMEAR	Pap Smear	REID'S SCORE	Reid's Score (Qualitative)	SWEDE'S SCORE	Swede's Score (Qualitative)
1	NEGATIVE	Negative	2	Negative	4	Negative
2	NEGATIVE	Negative	2	Negative	3	Negative
3	NEGATIVE	Negative	2	Negative	3	Negative
4	NEGATIVE	Negative	3	Positive	5	Positive
5	NEGATIVE	Negative	3	Positive	4	Negative
6	LSIL	Positive	4	Positive	7	Positive
7	NEGATIVE	Negative	0	Negative	0	Negative
8	NEGATIVE	Negative	2	Negative	4	Negative
9	NEGATIVE	Negative	2	Negative	3	Negative
10	NEGATIVE	Negative	2	Negative	3	Negative
11	NEGATIVE	Negative	2	Negative	3	Negative
12	NEGATIVE	Negative	2	Negative	3	Negative
13	NEGATIVE	Negative	1	Negative	2	Negative
14	NEGATIVE	Negative	2	Negative	3	Negative
15	NEGATIVE	Negative	2	Negative	3	Negative
16	NEGATIVE	Negative	2	Negative	3	Negative

17	NEGATIVE	Negative	2	Negative	3	Negative
18	NEGATIVE	Negative	2	Negative	4	Negative
19	LSIL	Positive	4	Positive	6	Positive
20	NEGATIVE	Negative	2	Negative	3	Negative
21	NEGATIVE	Negative	2	Negative	2	Negative
22	NEGATIVE	Negative	2	Negative	2	Negative
23	NEGATIVE	Negative	2	Negative	3	Negative
24	NEGATIVE	Negative	2	Negative	3	Negative
25	NEGATIVE	Negative	2	Negative	4	Negative
26	NEGATIVE	Negative	2	Negative	4	Negative
27	NEGATIVE	Negative	2	Negative	4	Negative
28	NEGATIVE	Negative	2	Negative	4	Negative
29	NEGATIVE	Negative	2	Negative	4	Negative
30	NEGATIVE	Negative	2	Negative	3	Negative
31	NEGATIVE	Negative	2	Negative	4	Negative
32	NEGATIVE	Negative	2	Negative	4	Negative
33	NEGATIVE	Negative	2	Negative	4	Negative
34	NEGATIVE	Negative	2	Negative	4	Negative
35	NEGATIVE	Negative	2	Negative	4	Negative
36	NEGATIVE	Negative	2	Negative	3	Negative
37	HSIL	Positive	6	Positive	7	Positive
38	NEGATIVE	Negative	1	Negative	2	Negative
39	NEGATIVE	Negative	2	Negative	4	Negative
40	NEGATIVE	Negative	1	Negative	3	Negative
41	NEGATIVE	Negative	2	Negative	3	Negative
42	NEGATIVE	Negative	2	Negative	4	Negative
43	NEGATIVE	Negative	2	Negative	4	Negative
44	NEGATIVE	Negative	2	Negative	3	Negative
45	NEGATIVE	Negative	1	Negative	3	Negative
46	NEGATIVE	Negative	2	Negative	4	Negative
47	NEGATIVE	Negative	1	Negative	3	Negative
48	NEGATIVE	Negative	2	Negative	4	Negative
49	NEGATIVE	Negative	2	Negative	4	Negative
50	LSIL	Positive	3	Positive	6	Positive
51	NEGATIVE	Negative	2	Negative	3	Negative
52	LSIL	Positive	3	Positive	6	Positive
53	LSIL	Positive	4	Positive	7	Positive
54	NEGATIVE	Negative	2	Negative	4	Negative
55	NEGATIVE	Negative	2	Negative	3	Negative
56	NEGATIVE	Negative	2	Negative	3	Negative
57	NEGATIVE	Negative	2	Negative	4	Negative
58	NEGATIVE	Negative	2	Negative	3	Negative
59	NEGATIVE	Negative	2	Negative	4	Negative
60	LSIL	Positive	3	Positive	6	Positive
61	NEGATIVE	Negative	2	Negative	4	Negative
62	NEGATIVE	Negative	2	Negative	3	Negative

63	NEGATIVE	Negative	2	Negative	4	Negative
64	NEGATIVE	Negative	2	Negative	4	Negative
65	NEGATIVE	Negative	2	Negative	4	Negative
66	NEGATIVE	Negative	2	Negative	4	Negative
67	HSIL	Positive	5	Positive	7	Positive
68	NEGATIVE	Negative	3	Positive	4	Negative
69	NEGATIVE	Negative	2	Negative	4	Negative
70	NEGATIVE	Negative	2	Negative	3	Negative
71	NEGATIVE	Negative	2	Negative	4	Negative
72	NEGATIVE	Negative	2	Negative	4	Negative
73	NEGATIVE	Negative	2	Negative	3	Negative
74	HSIL	Positive	4	Positive	6	Positive
75	NEGATIVE	Negative	2	Negative	3	Negative
76	NEGATIVE	Negative	2	Negative	3	Negative
77	NEGATIVE	Negative	2	Negative	4	Negative
78	NEGATIVE	Negative	2	Negative	3	Negative
79	LSIL	Positive	3	Positive	6	Positive
80	LSIL	Positive	3	Positive	7	Positive
81	HSIL	Positive	6	Positive	7	Positive
82	NEGATIVE	Negative	2	Negative	4	Negative
83	NEGATIVE	Negative	2	Negative	4	Negative
84	NEGATIVE	Negative	2	Negative	4	Negative
85	NEGATIVE	Negative	2	Negative	4	Negative
86	LSIL	Positive	3	Positive	6	Positive
87	NEGATIVE	Negative	2	Negative	4	Negative
88	NEGATIVE	Negative	2	Negative	5	Positive
89	LSIL	Positive	4	Positive	8	Positive
90	LSIL	Positive	3	Positive	7	Positive
91	NEGATIVE	Negative	2	Negative	4	Negative
92	NEGATIVE	Negative	2	Negative	3	Negative
93	NEGATIVE	Negative	2	Negative	4	Negative
94	NEGATIVE	Negative	2	Negative	4	Negative
95	LSIL	Positive	4	Positive	7	Positive
96	LSIL	Positive	4	Positive	7	Positive
97	LSIL	Positive	4	Positive	7	Positive
98	NEGATIVE	Negative	2	Negative	4	Negative
99	NEGATIVE	Negative	2	Negative	4	Negative
100	NEGATIVE	Negative	2	Negative	4	Negative
101	NEGATIVE	Negative	2	Negative	4	Negative
102	NEGATIVE	Negative	2	Negative	3	Negative
103	NEGATIVE	Negative	2	Negative	3	Negative
104	NEGATIVE	Negative	3	Positive	5	Positive
105	NEGATIVE	Negative	3	Positive	4	Negative
106	LSIL	Positive	4	Positive	6	Positive
107	NEGATIVE	Negative	0	Negative	0	Negative
108	NEGATIVE	Negative	2	Negative	4	Negative

109	NEGATIVE	Negative	2	Negative	3	Negative
110	NEGATIVE	Negative	2	Negative	3	Negative
111	NEGATIVE	Negative	2	Negative	3	Negative
112	NEGATIVE	Negative	2	Negative	3	Negative
113	NEGATIVE	Negative	1	Negative	2	Negative
114	NEGATIVE	Negative	2	Negative	3	Negative
115	NEGATIVE	Negative	2	Negative	3	Negative
116	NEGATIVE	Negative	2	Negative	3	Negative
117	NEGATIVE	Negative	2	Negative	3	Negative
118	NEGATIVE	Negative	2	Negative	4	Negative
119	LSIL	Positive	4	Positive	6	Positive
120	NEGATIVE	Negative	2	Negative	3	Negative
121	HSIL	Positive	6	Positive	7	Positive
122	HSIL	Positive	6	Positive	7	Positive
123	NEGATIVE	Negative	2	Negative	3	Negative
124	NEGATIVE	Negative	2	Negative	3	Negative
125	NEGATIVE	Negative	2	Negative	4	Negative
126	NEGATIVE	Negative	2	Negative	4	Negative
127	NEGATIVE	Negative	2	Negative	4	Negative
128	NEGATIVE	Negative	2	Negative	4	Negative
129	NEGATIVE	Negative	2	Negative	4	Negative
130	NEGATIVE	Negative	2	Negative	3	Negative
131	NEGATIVE	Negative	2	Negative	4	Negative
132	NEGATIVE	Negative	2	Negative	4	Negative
133	NEGATIVE	Negative	2	Negative	4	Negative
134	NEGATIVE	Negative	2	Negative	4	Negative
135	NEGATIVE	Negative	2	Negative	4	Negative
136	NEGATIVE	Negative	2	Negative	3	Negative
137	NEGATIVE	Negative	2	Negative	3	Negative
138	NEGATIVE	Negative	1	Negative	2	Negative
139	NEGATIVE	Negative	2	Negative	4	Negative
140	HSIL	Positive	6	Positive	8	Positive
141	NEGATIVE	Negative	2	Negative	3	Negative
142	NEGATIVE	Negative	2	Negative	4	Negative
143	NEGATIVE	Negative	2	Negative	4	Negative
144	NEGATIVE	Negative	2	Negative	3	Negative
145	NEGATIVE	Negative	1	Negative	3	Negative
146	NEGATIVE	Negative	2	Negative	4	Negative
147	NEGATIVE	Negative	1	Negative	3	Negative
148	NEGATIVE	Negative	2	Negative	4	Negative
149	NEGATIVE	Negative	2	Negative	4	Negative
150	LSIL	Positive	4	Positive	6	Positive
151	LSIL	Positive	4	Positive	6	Positive
152	NEGATIVE	Negative	2	Negative	3	Negative
153	HSIL	Positive	6	Positive	8	Positive
154	LSIL + BACTERIAL VAGINOSIS	Positive	4	Positive	6	Positive

155	LSIL + BACTERIAL VAGINOSIS	Positive	4	Positive	6	Positive
156	ASCUS	Positive	5	Positive	7	Positive
157	NEGATIVE	Negative	2	Negative	4	Negative
158	HSIL	Positive	7	Positive	9	Positive
159	NEGATIVE	Negative	2	Negative	4	Negative
160	LSIL	Positive	4	Positive	6	Positive
161	LSIL	Positive	4	Positive	6	Positive
162	NEGATIVE	Negative	2	Negative	4	Negative
163	NEGATIVE	Negative	2	Negative	4	Negative
164	NEGATIVE	Negative	2	Negative	4	Negative
165	NEGATIVE	Negative	2	Negative	4	Negative
166	NEGATIVE	Negative	2	Negative	3	Negative
167	LSIL	Positive	4	Positive	6	Positive
168	LSIL	Positive	4	Positive	6	Positive
169	LSIL	Positive	4	Positive	6	Positive
170	NEGATIVE	Negative	2	Negative	4	Negative
171	NEGATIVE	Negative	2	Negative	4	Negative
172	NEGATIVE	Negative	2	Negative	4	Negative
173	NEGATIVE	Negative	2	Negative	4	Negative
174	NEGATIVE	Negative	4	Positive	6	Positive
175	NEGATIVE	Negative	2	Negative	4	Negative
176	NEGATIVE	Negative	2	Negative	4	Negative
177	LSIL	Positive	4	Positive	6	Positive
178	LSIL	Positive	4	Positive	6	Positive
179	LSIL	Positive	4	Positive	6	Positive
180	NEGATIVE	Negative	4	Positive	6	Positive
181	LSIL	Positive	4	Positive	6	Positive
182	LSIL	Positive	4	Positive	6	Positive
183	NEGATIVE	Negative	2	Negative	3	Negative
184	LSIL	Positive	4	Positive	6	Positive
185	LSIL	Positive	4	Positive	6	Positive
186	NEGATIVE	Negative	2	Negative	4	Negative
187	NEGATIVE	Negative	2	Negative	3	Negative
188	HSIL	Positive	7	Positive	8	Positive
189	NEGATIVE	Negative	2	Negative	4	Negative
190	NEGATIVE	Negative	2	Negative	3	Negative
191	NEGATIVE	Negative	2	Negative	3	Negative
192	NEGATIVE	Negative	2	Negative	3	Negative
193	NEGATIVE	Negative	2	Negative	4	Negative
194	NEGATIVE	Negative	2	Negative	4	Negative
195	NEGATIVE	Negative	2	Negative	4	Negative
196	NEGATIVE	Negative	2	Negative	3	Negative
197	HSIL	Positive	5	Positive	6	Positive
198	NEGATIVE	Negative	2	Negative	4	Negative
199	NEGATIVE	Negative	2	Negative	5	Positive
200	NEGATIVE	Negative	2	Negative	4	Negative

201	NEGATIVE	Negative	2	Negative	3	Negative
202	NEGATIVE	Negative	2	Negative	4	Negative
203	NEGATIVE	Negative	2	Negative	3	Negative
204	LSIL	Positive	3	Positive	7	Positive
205	NEGATIVE	Negative	2	Negative	2	Negative
206	LSIL	Positive	4	Positive	8	Positive
207	NEGATIVE	Negative	2	Negative	2	Negative
208	NEGATIVE	Negative	2	Negative	4	Negative
209	NEGATIVE	Negative	2	Negative	3	Negative
210	NEGATIVE	Negative	2	Negative	2	Negative
211	NEGATIVE	Negative	2	Negative	2	Negative
212	NEGATIVE	Negative	2	Negative	3	Negative
213	NEGATIVE	Negative	2	Negative	3	Negative
214	NEGATIVE	Negative	2	Negative	2	Negative
215	NEGATIVE	Negative	1	Negative	3	Negative
216	NEGATIVE	Negative	2	Negative	3	Negative
217	NEGATIVE	Negative	2	Negative	2	Negative
218	HSIL	Positive	5	Positive	6	Positive
219	NEGATIVE	Negative	2	Negative	3	Negative
220	NEGATIVE	Negative	3	Positive	4	Negative
221	NEGATIVE	Negative	2	Negative	3	Negative
222	NEGATIVE	Negative	2	Negative	3	Negative
223	NEGATIVE	Negative	1	Negative	3	Negative
224	NEGATIVE	Negative	2	Negative	3	Negative
225	NEGATIVE	Negative	2	Negative	3	Negative
226	NEGATIVE	Negative	2	Negative	2	Negative
227	NEGATIVE	Negative	2	Negative	2	Negative
228	NEGATIVE	Negative	1	Negative	2	Negative
229	NEGATIVE	Negative	2	Negative	3	Negative
230	NEGATIVE	Negative	2	Negative	3	Negative
231	NEGATIVE	Negative	2	Negative	3	Negative
232	NEGATIVE	Negative	1	Negative	3	Negative
233	NEGATIVE	Negative	2	Negative	4	Negative
234	NEGATIVE	Negative	2	Negative	3	Negative
235	NEGATIVE	Negative	3	Positive	5	Positive
236	NEGATIVE	Negative	2	Negative	4	Negative
237	HSIL	Positive	6	Positive	7	Positive
238	NEGATIVE	Negative	2	Negative	2	Negative
239	NEGATIVE	Negative	2	Negative	2	Negative
240	NEGATIVE	Negative	2	Negative	4	Negative
241	NEGATIVE	Negative	2	Negative	3	Negative
242	NEGATIVE	Negative	2	Negative	3	Negative
243	NEGATIVE	Negative	1	Negative	3	Negative
244	NEGATIVE	Negative	2	Negative	2	Negative
245	NEGATIVE	Negative	2	Negative	2	Negative
246	NEGATIVE	Negative	2	Negative	3	Negative

247	NEGATIVE	Negative	2	Negative	3	Negative
248	NEGATIVE	Negative	3	Positive	5	Positive
249	NEGATIVE	Negative	2	Negative	2	Negative
250	LSIL	Positive	4	Positive	7	Positive
251	LSIL	Positive	3	Positive	6	Positive
252	NEGATIVE	Negative	2	Negative	3	Negative
253	NEGATIVE	Negative	2	Negative	3	Negative
254	NEGATIVE	Negative	2	Negative	2	Negative
255	NEGATIVE	Negative	2	Negative	2	Negative
256	LSIL	Positive	4	Positive	7	Positive
257	NEGATIVE	Negative	2	Negative	4	Negative
258	HSIL	Positive	5	Positive	7	Positive
259	NEGATIVE	Negative	2	Negative	2	Negative
260	LSIL	Positive	4	Positive	7	Positive
261	LSIL	Positive	4	Positive	6	Positive
262	NEGATIVE	Negative	2	Negative	2	Negative
263	NEGATIVE	Negative	2	Negative	4	Negative
264	NEGATIVE	Negative	2	Negative	4	Negative
265	LSIL	Positive	4	Positive	6	Positive
266	NEGATIVE	Negative	2	Negative	3	Negative
267	LSIL	Positive	4	Positive	7	Positive
268	LSIL	Positive	4	Positive	6	Positive
269	LSIL	Positive	5	Positive	8	Positive
270	NEGATIVE	Negative	2	Negative	4	Negative
271	NEGATIVE	Negative	2	Negative	4	Negative
272	NEGATIVE	Negative	2	Negative	4	Negative
273	NEGATIVE	Negative	2	Negative	4	Negative
274	LSIL	Positive	4	Positive	6	Positive
275	NEGATIVE	Negative	2	Negative	4	Negative
276	NEGATIVE	Negative	2	Negative	4	Negative
277	LSIL	Positive	4	Positive	7	Positive
278	LSIL	Positive	3	Positive	6	Positive
279	LSIL	Positive	5	Positive	7	Positive
280	NEGATIVE	Negative	2	Negative	3	Negative
281	LSIL	Positive	4	Positive	7	Positive
282	LSIL	Positive	3	Positive	6	Positive
283	NEGATIVE	Negative	2	Negative	4	Negative
284	LSIL	Positive	3	Positive	6	Positive
285	LSIL	Positive	4	Positive	7	Positive
286	NEGATIVE	Negative	2	Negative	3	Negative
287	NEGATIVE	Negative	2	Negative	3	Negative
288	NEGATIVE	Negative	2	Negative	3	Negative
289	NEGATIVE	Negative	2	Negative	4	Negative
290	NEGATIVE	Negative	2	Negative	2	Negative
291	NEGATIVE	Negative	2	Negative	2	Negative
292	NEGATIVE	Negative	1	Negative	2	Negative

293	NEGATIVE	Negative	2	Negative	4	Negative
294	NEGATIVE	Negative	2	Negative	4	Negative
295	NEGATIVE	Negative	1	Negative	3	Negative
296	NEGATIVE	Negative	2	Negative	4	Negative
297	NEGATIVE	Negative	2	Negative	3	Negative
298	NEGATIVE	Negative	2	Negative	4	Negative
299	NEGATIVE	Negative	2	Negative	2	Negative
300	NEGATIVE	Negative	1	Negative	3	Negative